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Pulmonary Embolism

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The importance of pulmonary embolism in clinical medicine is enhanced by the increasing population age and by better diagnosis and control of common pulmonary infectious disorders. Improvement in diagnostic techniques, together with the advent of anticoagulants also have increased interest in this condition.

Cecil and Loeb[1] believe pulmonary embolism is found in approximately 10 per cent of all autopsies and causes two to three per cent of all deaths. Castleman[2] has found that 90 per cent of all cardiac patients and 58 per cent of all postoperative patients autopsied demonstrated pulmonary emboli. Many were only incidental findings; in others, emboli contributed to or caused the patient's death.

The incidence of fatal pulmonary embolism actually is higher in nonsurgical than in surgical patients[3]. Approximately 30,000 deaths annually are estimated to occur from pulmonary embolism[4]. The real significance of this is better appreciated if we compare the 12,000 deaths from tuberculosis in the United States and the 40,000 people who lost their lives in automobile accidents in 1956. A recent study at the Graduate Hospital in Philadelphia disclosed that 60 pulmonary emboli occur per year in their hospital, considerably exceeding pneumonia (33 per year) or bronchogenic carcinoma (30 per year). Our greater awareness of this problem and our more extensive operations also contribute to the rise of this disease in modern hospitals. The figures on the incidence vary widely and the medical literature on all facets of the subject is confusing and contradictory as pointed out by DeBakey[6].

PREDISPOSING FACTORS

Pulmonary embolism is more common in older patients where it frequently is associated with heart disease, neoplasms, and major surgical procedures[5]. Inactivity, especially bed rest, numerous infections, subacute bacterial endocarditis, auricular fibrillation, and thromboangiitis obliterans also predispose to venous thrombosis and pulmonary embolism. In younger patients it often is the result of minor injuries or fractures.

This disease may precipitate or complicate heart failure; it frequently complicates myocardial infarction[7,8]. Operations on the pelvis or laparotomies with extensive resection of tissue especially predispose the patient to the development of venous thrombosis and pulmonary embolism. Allen, Barker, and Hines[9] feel that pulmonary embolism also occurs especially in the puerperium and in those over 40 as well as in patients with cancer, varicosities, previous history of thromboembolism, severe injuries, obesity, polycythemia, anemia, or infectious diseases. In one study of age in this disease it was found that 82 per cent of patients with thromboembolic disease were over 40[10]. Other factors deemed important in predisposing to pulmonary embolism are: occlusive arterial disease, enlarged hearts, and general debilitation. Approximately 90 per cent

of pulmonary emboli will occur in a predictable 30 per cent of patients[11]. Prolonged sitting, especially in a relatively cramped position, as well as the inactivity of the hemiplegic patient are predisposing factors in pulmonary embolism. No discernible predisposing factor can be found in approximately 6.5 per cent of cases[12].

In summary, factors predisposing to pulmonary embolism are[13]:

Preventable or treatable:

1 — Obesity
2 — Varicose veins
3 — Anemia
4 — Polycythemia
5 — Dehydration
6 — Shock
7 — Heart Failure
8 — Infectious Disease

Conditions that cannot be changed directly:

1 — Advancing age
2 — Bed rest
3 — Previous venous thrombosis
4 — Peripheral arterial insufficiency
5 — Pregnancy (in the puerperium)
6 — Surgical operations
7 — Injuries
8 — Burns
9 — Cancer
10 — Myocardial infarction

CAUSATIVE FACTORS

The causes of venous thrombosis and the possible resultant pulmonary embolism are generally considered to be: venous retardation; intimal vessel wall damage; and physiologic alterations in blood coagulability. Probably venous retardation is the most important factor. Incipient thrombi tend to be initiated at the apices of the valve pockets or may originate in the cul de sacs formed by the valve pockets where the venous stasis is maximal[12].

Knisley and Warner[14] have demonstrated blood sludging in special studies with the blood cells agglutinating into masses that settle to the lower side of the vessels and cement together, forming larger masses. Changes in blood convertin in certain experimental studies, in pregnancy, and in the early puerperium have been demonstrated[15]. A temporary rise in platelet adhesiveness also has been demonstrated postoperatively. Liberation of thromboplastic elements from gravid uteri, traumatized tissues, or necrotic tumors may contribute to the hypercoagulable state. Clot formation then can be initiated and potentiated by a degree of venous stasis that would not of itself result in thrombus formation.

PATHOLOGY

Venous thrombosis usually originates in the plantar veins or branches of the anterior and posterior tibial vessels of the foot and low calf, at least partly due to endothelial anoxemia there[10,16]. The older idea of separating thrombophlebitis and phlebothrombosis because of a supposed difference in their embolic potential appears not to be warranted[12,17]. Pulmonary emboli may occur in either condition. Approximately 10 per cent of pulmonary emboli arise in veins with what is considered to be thrombophlebitis[2] and 80 per cent of emboli in noncardiac cases come from phlebothrombosis in the extremities. In cardiac cases about 25 per cent of pulmonary emboli come from intramural cardiac thrombi, chiefly right atrial[1]. Arterial spasm may be so severe and profound in the presence of even unsuspected leg vein thrombosis as to lead to gangrene of the leg[18]. The leg veins probably initiate 90 per cent of the phlebothrombotic episodes[19]. Part of the disagreement in the literature on the primary venous thrombosis site undoubtedly arises because so few dissections are carried out below the inguinal ligaments.

The diseased or congested lung is much more likely to develop the full picture of pulmonary infarction after pulmonary embolism[2,20]. The full clinical picture of pulmonary infarction follows embolism in 90 per cent of patients with heart disease but in 60 per cent of patients with other diseases, presumably because of congestion and the slowed pulmonary circulation in patients with heart disease[8]. Pulmonary infarction develops against a pleural surface, especially in the costophrenic angles. It is probable that there is recanalization of the embolus later in many cases. On gross examination of a lung with pulmonary infarction, small fat tabs on the pleura give a clew to healed or healing pulmonary infarcts. Pulmonary emboli may give rise to hemorrhagic pleural effusion or, still later, to old linear fibrotic shadows on chest X-rays. Pulmonary infarction may lead to pulmonary cavitation[21]. Patients diagnosed as primary pulmonary artery thrombosis may actually have pulmonary artery thrombosis superimposed upon a previous embolus[17,22].

Approximately a decade ago, Lushbaugh and Steiner found widespread embolism of small pulmonary arteries, arterioles, and capillaries by the particulate matter found in the amniotic fluid and meconium in cases of obstetrical shock and death[23].

SYMPTOMS AND SIGNS

Venous thrombosis, in its early stages, may give remarkably few symptoms. Later, pain in the affected parts, most commonly pain in the foot, behind the ankle, and in the calf, especially with weight bearing, will be the earliest complaints. Slight foot or ankle swelling may occur. In some cases, the associated severe arterial spasm may give rise to blanching and even coldness in the affected limb.

On examination, swelling and tenderness on lateral compression of the low calf can be elicited in early venous thrombosis. Somewhat later, limitation of dorsiflexion and aggravation of the patient's calf pain on dorsiflexion of the foot may be elicited. Shock, congestive failure, or unexplained fever may be the only indication of pulmonary infarction[5]. Chest pain and frank venous thrombosis occurred in only 20 per cent of cases in one recent study[5]. Probably 75 per cent of fatal pulmonary emboli give some premonitory signs and symptoms[19].

With pulmonary embolism the obstruction between the two ventricles can produce three types of symptoms: insufficient cardiac output with peripheral circulatory failure and shock; signs and symptoms of pulmonary infarction; or signs of right ventricular failure (distended cervical neck veins and hepatomegaly)[24].

Insufficient Cardiac Output: Reduced cardiac output may be manifested by lowered blood pressure, clammy moist skin, increased pulse, decreased renal function, and elevation of the blood NPN. Syncope, often a presenting complaint for neurologic entities, may be due to the reduced left ventricular output with pulmonary infarction. Two reported cases had syncope without pain[5].

Signs and Symptoms of Pulmonary Infarctions: An increasing number of studies show that hemoptysis is comparatively rare[25] with pulmonary infarction even though the latter probably is the commonest cause of hemoptysis in nontuberculous patients. Fever may occur in pulmonary infarction; pain usually is absent unless the circulation is almost completely blocked. Symptoms and signs of pleurisy, consolidation, congestive failure, or plate-like areas of atelecatsis at the lung bases may occur. With the increasing awareness of the many bizarre syndromes that may originate from pulmonary infarction it is clear that hemoptysis, pain, or significant X-ray changes may all be absent[8].

The pain of pulmonary infarction may be pleurisy-like in character; substernal-like myocardial anoxia (when the embolism occurs in the larger branches of the pulmonary artery, this may be due to the associated diminished coronary artery flow); or pain from induced myocardial infarction, which may occur with pulmonary embolism even with normal coronary arteries.

In a recent article, Silverman[26] points out that pulmonary embolism, like many other types of cardiovascular disease, on careful bedside examination, will show important differentiating features, largely those of pulmonary hypertension. He refers especially to a heaving pulsation in the left lower parasternal area, systolic lifting in the second left intercostal space adjacent to the sternum, a palpable shock in the pulmonic area, and a conspicuous presystolic jugular pulsation. Localized chest wall tenderness, intense cyanosis, or hemoptysis favor pulmonary embolism.

Signs of Right Ventricular Failure: An accentuated pulmonic second sound, progressive dypsnea, and a full-blown picture of cor pulmonale may be the presenting symptoms of patients with pulmonary embolism[8].

DIAGNOSIS

Pulmonary infarction may take bizarre patterns and mimic many other diseases. There are no absolute diagnostic criteria, so that an accumulation of circumstantial evidence must be relied upon to make the diagnosis. The classical clinical and laboratory features of pulmonary embolism seldom are encountered[5].

Clincial Evidence: Important signs of venous thrombosis on physical examination are low calf or plantar tenderness, temperature difference in the two legs, a difference in the vein size of the two legs, and difference in the circumference of the calves[8]. An unexplained rise in the temperature and pulse may be due to an otherwise

asymptomatic venous thrombosis or pulmonary embolism.

X-ray: In 55 per cent of patients with pulmonary embolism, informed radiologists may suggest this possibility from X-ray studies[5,20]. The most common X-ray findings are linear markings, not the typical wedge shadow. Approximately 24 to 36 hours must elapse, following pulmonary embolism, before X-rays will give their first evidences of pulmonary infarction[2]. The X-ray pattern found in pulmonary embolism may be of several types[15]. With pulmonary infarction — a truncated cone with a base on one or more pleural surfaces (costal, diaphragmatic, mediastinal, or interlobar) may appear; without pulmonary infarction—congestive heart failure, pulmonary edema, pleural effusion, respiratory inhibition (with a high diaphragm and limited excursion, decreased distance from the dome of the diaphragm to the horizontal interlobar fissure, areas of atelectasis, often basal and platelike), and pulmonary oligemia with increased translucency may be the picture.

Later, the chest X-ray picture of pulmonary hypertension may develop with dilatation and increased pulsation of the hilar arteries or chronic cor pulmonale. Here the heart resembles mitral stenosis without dilatation of the left atrium[15].

Electrocardiogram: Electrocardiographic abnormalities may be detected in 70 per cent of patients who are adequately examined[5]. Israel and Goldstein[5], in a group of 60 patients, found a picture of cor pulmonale in seven, transient positional changes in 28, and coronary insufficiency patterns in 18. Nevertheless, the electrocardiogram seldom is of great help because so many of the abnormalities noted are nonspecific. The electrocardiographic pattern of acute cor pulmonale was evident in 20 per cent of a large group studied by Carlotti and associates[27].

Serum Glutamic Oxalacetic Transaminase (SGOT): The value of SGOT determinations in the diagnosis of pulmonary embolism or in excluding similar clinical entities is still controversial[28,29]. In one study, patients with pulmonary infarction showing SGOT elevations tended to have the peak rise between the third and sixth day, with an average level of 79, whereas in acute myocardial infarction the mean peak was 26 hours after the onset of the attack and the level was much higher[28]. In animal studies this enzyme is highly concentrated in liver, kidney, brain, and heart muscle as compared to skeletal muscle. SGOT also may be elevated in obstructive jaundice, pancreatitis, rheumatic myocarditis, renal infarction, hepatic necrosis, and in diseases of the musculoskeletal system[5,30]. Most observers report normal SGOT values in patients with pulmonary infarction where other concomitant diseases do not exist.

That there is ample room for refinement of our diagnostic techniques in this disease is amply demonstrated by a study in which Miller and Berry[31] found only 42 per cent of fatal cases of pulmonary infarction correctly diagnosed clinically. The typical picture of pulmonary infarction was seen in only 15 of their 104 cases. In 14, none of the so-called classic findings were present — pleural pain, hemoptysis, dypsnea, or friction rub. Tachycardia, mild dypsnea, and reduced blood pressure should be warnings of possible manifestations of pulmonary embolism and infarction. Pulmonary embolism must be differentiated from coronary artery disease, postoperative pneumonia, pulmonary neoplasms, and tuberculosis. It also may present as pleurisy with effusion, abscess, empyema, or pneumonia. It may simulate an acute surgical abdomen, hepatitis, or neurologic disease with syncope and vertigo[5]. Various forms of chronic myocarditis must be differentiated from repeated small pulmonary emboli[8].

PROGNOSIS

The diagnosis of thrombophlebitis carries approximately a 5 per cent mortality[10]. Israel and Goldstein[5] found that following pulmonary emboli, 77 per cent of their patients recovered.

PREVENTION

The incidence of venous thrombosis and consequent pulmonary embolism can be reduced by eliminating the causative factors[15]: Intimal damage — avoid subcutaneous or intravenous injections in the lower extremities, dilute hypertonic or irritating solutions; venous stasis — avoid immobilization, use elastic supports on the legs; increase blood coagulability — administer anticoagulants.

Generally applicable prophylactic measures are essential if we are to make any progress on this problem. The incidence of pulmonary embolism will be reduced if the preoperative preparation includes reduction in the obese patient, correction of varicose veins, medical supervision of cardiac

abnormalities (congestive failure and arrythmia), and correction of anemia[32]. Phlebothrombosis can be anticipated in patients who are malnourished, with recent shock, acute coronary occlusion, mild congestive failure, lower abdominal surgery, the listless inactive patient, or those with anemia, carcinoma, or intra-abdominal inflammatory disease[16]. A program to prevent venous thrombosis should include[10] proper positioning of the patient on the operating table, avoiding damage to veins or pressure on extremities, postoperative elevation at the foot of the bed, breathing and leg exercises every hour, early ambulation, and avoiding heel-less slippers.

Avoiding Immobilization: Lowe et al.,[32] believe early ambulation is associated with a reduction in the incidence of thromboembolic disease from 53 to 18 per cent. Their studies further show that the incidence of pulmonary embolism is especially reduced with early ambulation. Many authors emphasize that early ambulation must be early, active, vigorous, repeated, and not "too little and too late" if it is to be effective in preventing venous thrombosis.

Elastic Stockings: Significant reduction of the incidence of pulmonary embolism has followed the routine application of elastic stockings for all immobilized patients[33,34]. Two similar sized groups, one serving as controls and the other wearing stockings, demonstrate this well on the following table:

	Stockings	Controls
Fatal pulmonary embolism	0	4
Nonfatal pulmonary embolism	2	12

The authors believe every hospital patient may safely wear these elastic stockings, except those with severe disease of the legs such as ischemia, inflammation, or trauma. Elastic stockings are not a substitute for but an addition to other measures designed to prevent pulmonary embolism. Not all investigators using the elastic leg support as a routine preventive procedure have found them useful[10].

Vein Ligation: Ochsner[3] urges prophylactic vein ligation on the cardiac side of the thrombus. Other authors feel bilateral superficial vein ligation should be used only if anticoagulant treatment is contraindicated or pulmonary emboli persist in the face of adequate anticoagulant therapy[10,13,17].

Anticoagulants: Many studies are reported in the literature advocating the use of anticoagulant drugs on a nearly routine basis in the early postoperative period for the prevention of venous thrombosis and pulmonary emboli[1,16]. Other studies have reported success in reducing the incidence of pulmonary emboli—using continous anticoagulant therapy in patients with rheumatic heart disease, auricular fibrillation, or severe chronic congestive failure[1]. Both heparin and Dicumarol® have been recommended for these purposes[32]. Stinchfield's[35] studies suggest that anticoagulants impair bone healing. It probably is preferable to avoid routine anticoagulant administration in patients with renal insufficiency, brain or spinal cord surgery, blood dyscrasias, ulcerative lesions, nutritional deficiency, or hepatic disease[36]. The value of routine postoperative anticoagulants in preventing pulmonary embolism in the especially predisposed was established more than ten years ago[36]:

	Expected	Occurred
832 with abdominal hysterectomy		
— venous thrombosis or pulmonary embolism	33	3*
— fatal pulmonary embolism	6	0

(* minor venous thrombosis)

The Mayo Clinic[40] now uses prophylactic anticoagulant therapy for postoperative patients who have had venous thrombosis or pulmonary embolism previously and for most patients who have pelvic laparotomies. Donaldson and associates[37] use anticoagulants in individuals over 40 years of age who undergo major surgery and are bound to be debilitated for two days or more. The prothrombin determination is maintained depressed until the patient is active and has regained normal leg muscle tone. Younger people especially predisposed to phlebitis (obese, intestinal obstruction, distention, or particularly debilitating surgery) also are given Dicumarol. Other institutions report success with similar programs[13,16].

TREATMENT

Vein Ligation: Bilateral high ligation of the superficial femoral veins has been advocated for thrombosis of the leg veins. Because of swelling of the legs, varicose veins, ulceration, and cramping that may develop, it appears to be losing favor

gradually[37]. Enthusiasm for ligating the vena cava also appears to be waning. The dypsnea and the postphlebitic syndrome that commonly develops with exercising the legs makes the value of the procedure dubious.

Anticoagulants: Refinements in the techniques of anticoagulant administration are making these preparations safer[38]. In a recent study of 5,000 patients receiving anticoagulants there were no deaths due to anticoagulants. Long term anticoagulant therapy has been successfully used in rheumatic heart disease with embolic episodes, myocardial infarction, and in patients with recurrent thrombophlebitis with or without pulmonary emboli[39]. The need for Dicumarol is decreased during an upper respiratory tract infection, diarrhea, intestinal sterilizing antibiotics, low food intake, or excess alcohol. Approximately 50 mg. per day will maintain most patients in the desirable 20 to 35 sec. prothrombin range. The value of this form of therapy in preventing thromboembolic episodes in patients with recurrent thrombophlebitis is seen in the following table[5].

Patients	Without Anticoagulants		With Anticoagulants	
	Pt. Mo.	Episodes	Pt. Mo.	Episodes
24	2207	92*	896	7**

*Includes 63 episodes of thrombophlebitis and 20 of pulmonary embolism

**Thrombophlebitis in 7 patients

SUMMARY

Because pulmonary embolism permeates nearly all fields of clinical medicine it is appropriate to review some of the new considerations for the detection and management of this common disorder. Untreated, it tends to progress to chronic cor pulmonale, severe peripheral venous insufficiency, or even sudden death. It is estimated that in this country 30,000 people per year die from thromboembolism. Its prevention or effective treatment is of prime importance.

For decades, the triad of intimal damage, venous stasis, and increased blood coagulability has been incriminated as causing phlebothrombosis and pulmonary infarction. Of these, venous stasis probably is most important. Factors contributing to the hypercoagulable state, according to recent studies, include a temporary rise in the convertin complex, increased platelet adhesiveness, and possible liberation of thromboplastic elements from a gravid uterus, traumatized tissues, or necrotic tumors.

In the absence of any positive tests for phlebothrombosis or pulmonary infarction, the diagnosis is best established with a careful history, a meticulous physical examination, and a high index of suspicion. All ancillary aids from the laboratory and X-ray departments constitute probable evidence that aids materially in establishing the diagnosis of pulmonary embolism and in excluding numerous similar clinical conditions. Clinical evidence, together with serial X-ray studies for the usual signs of pulmonary embolism (infarction, respiratory inhibition, basal atelectasis, pulmonary oligemia, dilatation of the hilar arteries, pleural effusion, or pulmonary edema) usually will permit a reliable differentiation from the various types of pneumonitis. In cases of suspected pulmonary infarction, where pneumonia cannot be excluded with reasonable certainty, it probably is preferable to include antibiotics in the treatment. Serial electrocardiograms and serial determinations of the serum glutamic oxalacetic acid transaminase level (SGOT), together with other available clinical information, usually permits the differentiation from myocardial infarction.

Early ambulation was associated with a reduction in the incidence of thromboembolic disease from 53 to 18 per cent in one carefully studied series. The routine use of low pressure elastic stockings in all immobilized patients appears to be effective in preventing venous thrombosis and pulmonary embolism. Efforts to avoid the immobilization that permits venous thrombosis to be initiated must be continued. A growing number of medical centers are using anticoagulants in a nearly routine fashion postoperatively in an effort to prevent venous thrombosis and possible pulmonary embolism. Many pulmonary emboli can be prevented by giving Dicumarol or similar drug to all patients with chronic, severe, congestive heart failure or to patients with rheumatic heart disease or auricular fibrillation who cannot be converted to a sinus rhythm.

Prompt and effective neutralization of the hypercoagulable state with anticoagulant therapy for at least three to six weeks is generally accepted as the preferable method of treating established venous thrombosis or pulmonary embolism. Rest and elevation of the legs remain important

older principles in the management of these diseases. Surgical vein ligation probably is best reserved for situations where anticoagulants are not feasible.

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REFERENCES

1. Cecil and Loeb: Textbook of Medicine, 9th ed. Philadelphia, W. B. Saunders, 1955.
2. Castleman, B.: Personal communication, June 1952.
3. Ochsner, A.: Venous Thrombosis, Illinois M. J. **108**:213, 1955.
4. Pratt, G. H.: Postthrombotic Syndrome, S. Clin. North America **33**:1229, 1953.
5. Israel, H. L., and Goldstein, F.: The Varied Clinical Manifestations of Pulmonary Embolism, Ann. Int. Med. **47**: 202, 1957.
6. DeBakey, M. E.: Collective Review: Critical Evaluation of Problem of Thromboembolism, Internat. Abstr. Surg. **98**:1-27, 1954.
7. Friedberg, C. K.: Diseases of the Heart, 2nd ed. Philadelphia, W. B. Saunders, 1956.
8. Harrison, T.: Thrombophlebitis. Principles of Internal Medicine, 3rd ed. New York, Blakiston, 1958.
9. Allen, Barker, and Hines: Peripheral Vascular Diseases, 2nd ed. Philadelphia, W. B. Saunders, 1955.
10. Donaldson, G. A.: Personal communication, July 1952.
11. Farmer, D. A., and Smithwick, R. H.: Thromboembolic Diseases: Discussion of Problem in Surgical Patients with Particular Reference to Fatal Embolus, Angiology **1**:291, 1950.
12. Byrne, J. J.: Phlebitis, New England J. Med. **253**:579. 1955.
13. Hussey, H. H.: Pulmonary Embolism, Current Therapy, Philadelphia, W. B. Saunders, 1957.
14. Knisley, M. H., and Warner, L.: Thrombosis and Embolism, Proc. International Conference Basal 1954, Basal, Benno Schwabe, 1955.
15. Wessler, S.; Cohen, S.; and Fleischner, F. G.: The Temporary Thrombotic State, New England J. Med. **254**:413, 1956.
16. Allen, J. G.: Phlebothrombosis and Thrombophlebitis, Current Therapy, Philadelphia, W. B. Saunders, 1957.
17. McLachlin, J., and Patterson, J. C.: Venous Thrombosis and Pulmonary Embolism, Surg. Gynec. & Obst. **93**:1, 1951.
18. Waller, J. V.: Venous Thrombosis Simulating Arterial Embolization with Major Gangrene, J.A.M.A. **165**:344, 1957.
19. Crane, C.: Deep Venous Thrombosis and Pulmonary Embolism, New England J. Med. **257**:147, 1957.
20. Littler, J.: Thromboembolism: Its Prophylaxis and Medical Treatment, M. Clin. North America **36**:5, 1952.
21. Coke, L. R., and Dundee, J. C.: Cavitation in Bland Infarcts of the Lung, Canad. M.A.J. **72**:907, 1955.
22. Ring, A., and Bakke, J. R.: Chronic Massive Pulmonary Artery Thrombosis, Ann. Int. Med. **43**:781, 1955.
23. Lushbaugh, C. C., and Steiner, P. E.: Additional Observations on Maternal Pulmonary Embolism by Amniotic Fluid, Am. J. Obst. & Gynec. **43**:833, 1942.
24. Myers, G. B., and Talmers, F. N.: The Electrocardiographic Diagnosis of Acute Myocardial Ischemia, Ann. Int. Med. **43**:361, 1955.
25. Gunderson, P.: Pulmonary Embolism, Nord. med. **53**:565, 1955.
26. Silverman, J. J.: Bedside Clews in the Diagnosis of Cardiovascular Disease, J.A.M.A. **166**:725, 1958.
27. Carlotti, J.; Hardy, I. B.; Linton, R. R.; and White, P. D.: Pulmonary Embolism in Medical Patients, J.A.M.A. **134**:1447, 1947.
28. Ostrow, H.; Palis, G. N.; and Evans, J. M.: SGOT in Pulmonary Embolization, Clinical Research Proc. **4**:155, 1956.
29. Denny, J. L.; McAuley, C. B.; Martin, H. E.; Ware, A. G.; and Segalove, M.: Evaluation of the Serum Glutamic Oxalacetic Aminopherase (Transaminase) Test, J.A.M.A., **161**:614, 1956.
30. Walsh, J. R.; Humoller, F. L.; and Gullick, F. G.: Serum Transaminase in Pulmonary Disease, Ann. Int. Med. **46**: 1105, 1957.
31. Miller, R., and Berry, J. B.: Pulmonary Infarction - A Frequently Missed Diagnosis, Am. J. M. Sc. **222**:197, 1951.
32. Lowe, L.; Berger, L.; and Lasser, R. T.: The Prevention of Thromboembolism, Angiology **2**:26, 1951.
33. Wilkins, R. W.; Mixter, G., Jr.; Stanton, J. R.; and Litter, J.: Elastic Stockings in the Prevention of Pulmonary Embolism: A Preliminary Report, New England J. Med. **246**:361, 1952.
34. Wilkins, R. W., and Stanton, J. R.: Elastic Stockings in the Prevention of Pulmonary Embolism, New England J. Med. **248**:1087, 1953.
35. Stinchfield, F.; Sankaran, R.; and Samilson, R.: The Effect of Anticoagulant Therapy on Bone Repair, J. Bone and Joint Surg. **38-A**:270, 1956.
36. Allen, E. V.; Hines, E. A., Jr.; Kvale, W.; and Barker, N. W.: The Use of Dicumarol as an Anticoagulant: Experience in 2,307 Cases, Ann. Int. Med. **27**:371, 1947.
37. Donaldson, G. A.: Personal communication, May 1957.
38. Ware, A. G., and Stragnell, R.: Anticoagulant Therapy: Elimination of Some Commonly Occurring Pitfalls, Ann. Int. Med. **46**:451, 1957.
39. Foley, W. T.; McDevitt, E.; Symons, C.; and Wright, I. S.: Further Experience with Long Term Anticoagulant Therapy. A.M.A. Arch. Int. Med. **95**:497, 1955.
40. Barker, N. W.: Personal communication, May 1957.

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Management of the Hypercholesterolemic Patient

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Hypercholesterolemia presents a problem to the investigator, to the patient, and to the clinician. The investigator realizes most acutely how incomplete is our knowledge of the relationships between blood lipids and atherosclerosis. He knows that caution must be exercised in interpreting experimental and epidemiological results. Most of all, he knows that any regimen for altering blood lipids must be evaluated in controlled studies to determine its ultimate effect on atherosclerosis. The patient reads in popular publications that coronary artery disease, the leading killer, is related to the level of cholesterol in the blood and that blood cholesterol is somehow related to the diet. Even the most carefully written articles are likely to be misunderstood by persons without medical training, particularly since such a complex subject does not lend itself to oversimplification. The clinician finds himself surrounded by a welter of inconclusive and sometimes contradictory evidence. On the other hand, he finds his patients eager for information and advice.

Let us focus for a moment on basic orientation of the clinician. First, he must distinguish whether a given study refers to blood lipids, atherosclerosis, or complications of atherosclerosis, such as myocardial infarction. Although these conditions may be related, they are not synonymous. Not every patient with atherosclerosis has abnormalities in blood lipids; and not every patient with arterial occlusion has atherosclerosis. Besides abnormalities in lipids, other factors in the production of atherosclerosis include clearing factors, structure of the arterial wall, respiration and lipogenesis in the arterial wall, arterial hypertension, and turbulence. All these factors are being studied, but their roles—like those of the lipids—are incompletely understood. Atherosclerosis represents only one of several factors in arterial occlusions; of the others, blood coagulability is particularly important because thrombosis is nearly always the final event in arterial occlusion and may occur in the absence of atherosclerosis.

Cholesterol was early implicated in the etiology of atherosclerosis because of the ease of determining its serum concentration, its presence in plaques, and the occurrence of premature, severe atherosclerosis in several disorders characterized by hypercholesterolemia. The relationship between hypercholesterolemia and atherosclerosis is statistical, which means that although it applies to large groups, it does not necessarily apply to small groups or to the individual—something worth remembering when talking to the patient.

The lack of a test for uncomplicated atherosclerosis has been one of the foremost problems for clinician and investigator alike. At present, atherosclerosis cannot be detected until arterial occlusion occurs or until narrowing is sufficient to cause symptoms of ischemia such as angina pectoris or intermittent claudication. Until such a test is devised, we must attempt to evaluate the ultimate effect of treatment for hypercholesterolemia by detecting the thrombotic complications of atherosclerosis rather than by measuring degrees of atherosclerosis or its total extent. To date, attempts to improve on the predictive value of the blood cholesterol determination have centered around fractionation of lipoprotein cholesterol or total lipids, with particular attention to the beta fraction or low density lipoprotein fractions, which appear to be most guilty in the formation of atheromata. A committee of prominent investigators, designated to evaluate ultracentrifuge data in a co-operative study, reported last year that ultracentrifuge

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techniques did not improve on the accuracy of serum cholesterol determinations in predicting the probability of atherosclerosis[1]. Whether the simpler electrophoretic determination or a chemical determination of the beta fraction will give information more useful than the total cholesterol level remains to be seen.

DISCOVERY OF HYPERCHOLESTEROLEMIC PATIENTS

While awaiting more definite information, the clinician must decide what advice to give his patients right now. The most conservative approach would be to offer no treatment except prevention or elimination of obesity, the one measure on which everyone agrees. With such a viewpoint, there would be no need to determine serum cholesterol levels. At the other extreme some workers advise determining the serum cholesterol annually in all patients. Most would agree that the determination should be done in persons likely to have hypercholesterolemia. Several clews may lead to the discovery of this abnormality:

1. Conditions known to be associated with hypercholesterolemia (diabetes mellitus, hypothyroidism, nephrosis, bilateral oöphorectomy.)
2. Occurrence of coronary disease or arteriosclerosis obliterans in the patient at an early age.
3. Family history of coronary disease and/or premature arteriosclerosis obliterans.
4. Xanthelasmata.
5. Xanthoma tuberosum.
6. Xanthoma tendinosum.
7. Arcus senilis (premature).

Many hypercholesterolemic patients offer none of these clews and may be discovered by routine screening tests of the blood.

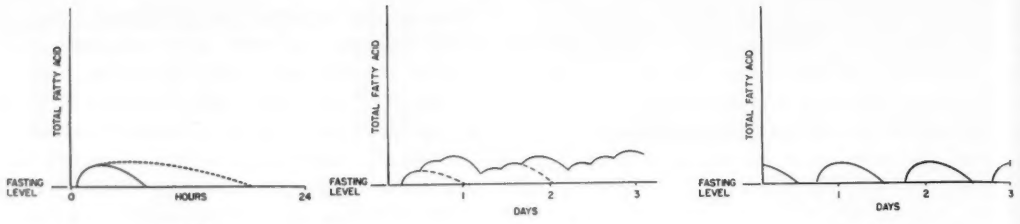
Our present view is that persons with any of these clews should be studied and that some form of treatment should be offered if hypercholesterolemia or elevation of the beta-lipoprotein cholesterol fraction is found. However, since there is no definite evidence that reduction in cholesterol levels is certain to lessen the chances of atherosclerosis or its complications, both physician and patient should understand that, regardless of the method of therapy, a clinical experiment is being conducted.

TYPES OF HYPERCHOLESTEROLEMIA

Although there has been much emphasis on exogenous factors, particularly diet, that influence cholesterol levels, the most important factor in determining the presence of hypercholesterolemia and its response to therapy is contained in the vague term, "the metabolic defect." As our understanding of hypercholesterolemia improves, it becomes apparent that it would be just as erroneous to lump together all forms of hypercholesterolemia as it would be to consider all forms of hyperglycemia as a single entity. Although the comparsion is not exact, it is convenient to think of hypercholesterolemia as a metabolic disorder analogous to diabetes. The most common form of hypercholesterolemia—called essential, primary, or familial—is a genetically determined, nonsex-linked defect sometimes associated with the presence of xanthelasmata, xanthoma tuberosum, and/or xanthoma tendinosum.

Secondary hypercholesterolemia occurs in a number of dissimilar situations including diabetes mellitus, nephrosis, hypothyroidism, and following bilateral oöphorectomy. It also is found in the condition known as essential familial hyperlipemia, in which the total lipid content of the serum is greatly elevated, chiefly in the neutral fat and fatty acid fractions, with secondary elevation of the cholesterol level. The plasma is grossly milky. Cause and treatment of this disorder differ from all other forms of hypercholesterolemia and have been so well described that we might well digress to consider them at this time.

When a normal individual ingests a meal containing fat, the total lipids in the blood respond in the manner illustrated in Figure 1a, with a return to normal in a few hours. The curve in a person with essential hyperlipemia takes a much longer time to return to normal (Figure 1a). Consequently, his next meal comes along before the level has returned to normal, with a gradual step-wise increase until extremely high levels are constantly present (Figure 1b). The disorder can be treated effectively by a program consisting of a fat-free breakfast and noon meal, with an evening meal containing whatever amount of fat is desired. This program of spaced fat feeding usually results in a higher than average intake of fat, since patients tend



(a) (b) (c)

Figure 1.—Familial hyperlipemia: (a) The solid line indicates a usual fat tolerance test in a normal individual, while the broken line indicates the delayed removal of fat found present in a person with familial hyperlipemia; (b) Here, shown diagrammatically, is the slow build-up of fat in the blood stream due to the fact that the fasting level is not reached before the next fat meal is ingested. The broken lines indicate how the removal of the fat from the blood stream might continue, had not another meal of fat been superimposed upon the elevated blood fat. What is shown here in exaggerated fashion continues over the years to build up hyperlipemia and hypercholesterolemia. (c) This shows, diagrammatically, when the spaced fat regimen is used, how the blood fats return to the fasting level before the next fat meal is ingested, even though the removal rate is prolonged.

Illustrations courtesy Annals of Internal Medicine (Wilkinson) **45**:*674-680 (October) 1956.*

to binge in the evenings. But hyperlipemia is avoided because ample time is permitted for return to normal before fat again is ingested (1c). Mild cases can be treated effectively by elimination of fat at breakfast only. In this condition, hypercholesterolemia disappears when hyperlipemia is controlled.

The exact nature of the metabolic defect that causes hypercholesterolemia has not been delineated. It is clear, however, that the serum cholesterol level is almost independent of dietary cholesterol. The mechanism regulating this level lies somewhere in the synthesis of cholesterol in the liver, or in the balance between the intestinal absorption and excretion of exogenous and biliary cholesterol. It is likely that the major defect lies in synthesis of cholesterol from the acetate pool and that absorption is of secondary significance. The rate of conversion of cholesterol to bile acids and their subsequent excretion in the feces also may be of considerable importance. Even though incompletely understood, it is apparent that the metabolic defect is of major importance and other factors, notably the dietary intake, are less significant in determining hypercholesterolemia. To assign the major role to dietary fat is akin to considering diabetes mellitus a disease caused by excessive intake of carbohydrates.

DIETARY TREATMENT: GENERAL

Treatment that might be offered to patients with hypercholesterolemia can be divided into dietary and nondietary methods. As mentioned, if we decide on dietary treatment, we do so not because diet causes hypercholesterolemia but because mild forms, like mild diabetes, sometimes can be controlled by diet. It is reasonably safe to say that in the human diet, only the fats and total calories are known to affect blood cholesterol levels. A review by Page et al.[2] offers an authoritative summary of the evidence regarding the role of dietary fat. In regard to the general population, they stated that the present evidence "does not suggest the desirability of any drastic dietary changes, specifically in the quantity or type of fat." On the other hand, they noted the deleterious effect of obesity (on arteriosclerosis, because of its increase in the work of the heart but not necessarily on hypercholesterolemia) and stated that "obesity is caused by consuming more energy than one expends, that dietary fats are the most concentrated source of energy, providing some 40 to 45 per cent of the daily caloric intake. Many should consume less calories, which — for most — means eating less fat. Diets providing 25 to 30 per cent of the calories from fat can still provide palatable meals for our accustomed tastes . . . The fat content should be sufficient only to meet caloric and essential fatty acid demands . . . These conclusions obviously apply to the general population and not to patients or to individuals with a strong family history of early death from cardiovascular disease, who are being observed with some regularity by their physicians. Here, the newer concepts of nutrition readily suggest various types of diet therapy that may prove useful to certain patients." This remark might

be construed as agreeing with the concept of offering therapy to any patient with hypercholesterolemia as a clinical experiment.

Restriction of dietary cholesterol, historically the first form of dietary treatment attempted, is ineffective, as already discussed. Restriction of total fat might be recommended because of the reported association of high fat intake with hypercholesterolemia. It is difficult, however, to disentangle fat content from other dietary and metabolic factors, including caloric balance, changes in body weight, and exercise. An undetermined number of the milder forms of hypercholesterolemia will be helped by sufficient restriction of fat. The critical point here, as in deciding on the clinical usefulness of each method of therapy, is in the degree of restriction required and the practicability of following the program indefinitely at home. No form of therapy is presently available that is effective unless employed continuously. Favorable results of short-term studies in a metabolic unit or in brief outpatient experiments do not necessarily indicate that a particular method is practical for clinical use.

UNSATURATED FATTY ACIDS

Many studies in the past few years have demonstrated that serum cholesterol can be reduced to some extent by substituting unsaturated vegetable fats for some of the saturated fats of the diet. The most successful programs have been the formula diets such as those used by Ahrens and associates and by Kinsell and associates. However, drinking three milk shakes daily instead of eating solid foods is not a way of life. Definite but less impressive results have been obtained when unsaturated fatty acids have been added to a more usual diet restricted in saturated fats. All of the reported studies covered relatively short periods of time. Persons with normal cholesterol levels and those with a slight metabolic defect (not synonymous with slight elevation of the serum level) may respond to dietary methods for reduction of circulating cholesterol; those with a more severe metabolic defect respond in a lesser degree or not at all. Once again, the main question is whether effective reduction of cholesterol levels can be achieved by a degree of dietary restriction that patients will follow indefinitely.

It seems clear that restriction of a given amount of saturated fat in the diet has about twice as much cholesterol-lowering effect as the addition of the same amount of unsaturated fat. Attempts to reduce serum cholesterol by adding unsaturated vegetable fats to an unrestricted diet have been unsuccessful generally.

One other fact worth noting is that the effect of corn oil, which is inexpensive, is approximately the same as that of preparations of safflower oil, which costs considerably more. The several pharmaceutical houses producing safflower oil preparations have seen fit to imply by name, by quotation of suggestive but inconclusive evidence, or by advertising innuendo that their preparations have value in preventing atherosclerosis. There is no definite evidence that this is true.

OTHER DIETARY METHODS

Another dietary method that could prove to be of limited value, if the results of one study can be duplicated, consists of the addition of lecithin in large doses to a diet low in fat (36 grams of lecithin daily and a 25 gram fat diet). Lesser dietary restriction and smaller doses of lecithin previously had given less impressive results in other studies. A French study has shown reduction in cholesterol following administration of a product combining 5 amino acids. To my knowledge, these results have not been confirmed in this country.

NONDIETARY TREATMENT

Nondietary methods of reducing serum cholesterol levels include the use of desiccated thyroid or its analogues, sitosterols, chelating agents, magnesium compounds, estrogens, and nicotinic acid.

A. THYROID AND THYROID ANALOGUES

The use of desiccated thyroid or its analogues results in reduction of elevated cholesterol levels when hypothyroidism is the cause of hypercholesterolemia. These products usually are ineffective when hypothyroidism is not the cause.

B. SITOSTEROLS

Sitosterols, plant sterols somewhat similar in structure to cholesterol, are said to bind cholesterol in the intestinal tract when ingested with food, resulting in greater fecal excretion and reduced absorption, with reduction of serum cholesterol levels. Most workers who have studied beta sitosterol have found it ineffective in hypercholesterolemia. The commercial product is expensive, large quantities are required, and its

failure in repeated studies suggests that it should not have a prominent place in clinical programs.

C. CHELATING AGENTS AND MAGNESIUM COMPOUNDS

Neither chelating agents nor magnesium compounds have any clinical value at present, although isolated reports have assigned cholesterol-reducing properties to each.

D. ESTROGENS

Estrogens in large doses are effective in lowering cholesterol levels, with reduction of the beta fraction and increase of the alpha fraction. In men, painful mammary enlargement and loss of sexual function preclude widespread clinical use of preparations currently available. Some important clinical studies are in progress, however, evaluating the effect on atherosclerosis in patients treated for many years and compared with controls. It is possible that a suitable weak estrogen may be developed, with the lipid effects but without significant sexual effect, in which case this could become a practical method of treatment. Caution must be used in determining the effect on endocrine balance and reproductive function when even weak estrogens are administered for long periods. At present, it is desirable to administer estrogens in physiologic or larger doses to oöphorectomized young women, in whom hypercholesterolemia and atherosclerosis are likely to develop in severe degree without such therapy.

E. NICOTINIC ACID

Nicotinic acid in large doses has been found in a number of studies in the past three years to be effective in reducing serum cholesterol levels. The first long-term study was one I helped initiate and the latest results of which were reported by my former associates at the Mayo Clinic.[3] Our study in Madison has yielded almost identical results. We now have 60 patients in the study and are continuing to expand it in order to insure long-term observation of a group large enough to assess the effect on the arteries. Our experience, in common with that of the Rochester group, the University of Saskatchewan group [where Altschul first suggested this method of treatment], and others, suggests that this is a safe and effective method of reducing serum cholesterol in the majority of hypercholesterolemic patients. Studies in cholesterol-fed rabbits by three independent groups have shown that nicotinic acid can prevent not only hypercholesterolemia but also aortic atherosclerosis. These results cannot be transferred to humans.

I shall summarize the results of our studies in 44 patients who have been followed for six to 24 months. In 18 who started in the second year of the study, lipoprotein cholesterol fractions have been determined as well as total cholesterol. Table 1 shows our usual dosage schedule. We start with 3 grams of nicotinic acid daily in divided doses after meals to minimize side effects. In general, the use of smaller doses has failed to give satisfactory results. If the serum cholesterol level is still above 250 mg% after 12 weeks at this level, the dose is increased progressively at 6-week intervals to 4.5 grams, 6 grams, 7.5 grams, and in a few cases to 9 grams, depending upon the response. To date we have found that doses higher than 6 grams a day do not improve on the effect of 6 grams in refractory cases. After 30 weeks, we substitute nicotinamide in equal dosage and have found that in every case, cholesterol levels have returned to the p r e t r e a t m e n t control range. Twelve weeks later, nicotinic acid is resumed and the dose further individualized if necessary. In the second year, therapy is discontinued for four weeks to permit each patient again to serve as his own control. Cholesterol levels return to

Table 1. Dosage Schedule

NICOTINIC ACID
0-12 weeks — 3 gm. daily (divided doses, p.c.)
12-30 weeks — Same dose if cholesterol normal.
If above 250 mg. % increased (4.5 gm., 6.0 gm., 7.5 gm. daily) at 6-8 week intervals.
30-42 weeks — NICOTINAMIDE in equal dosage.
42-56 weeks — Resume nicotinic acid and adjust dose further if necessary (9.0 gm. in few).
56-60 weeks — No treatment
60 weeks on — Resume NICOTINIC ACID

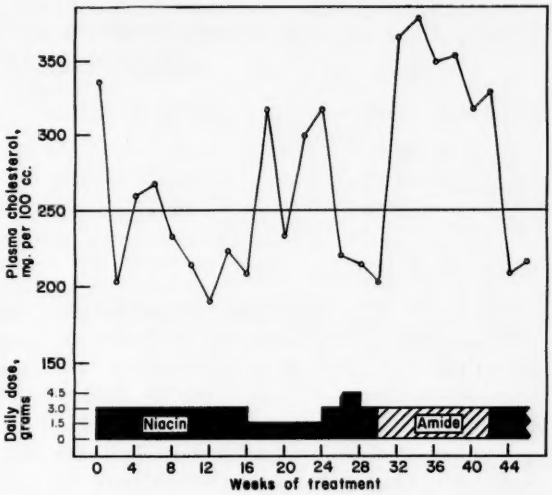


Figure 2.—Illustration of reduction of blood cholesterol levels by administration of 3 gm. of nicotinic acid daily, return to elevated levels during the use of 1.5 gm. of nicotinic acid daily, reduction to normal levels when a daily dose of 3 gm. of nicotinic acid was resumed, return to elevated levels when nicotinamide was substituted, and reduction to normal levels when the use of nicotinic acid was again resumed.

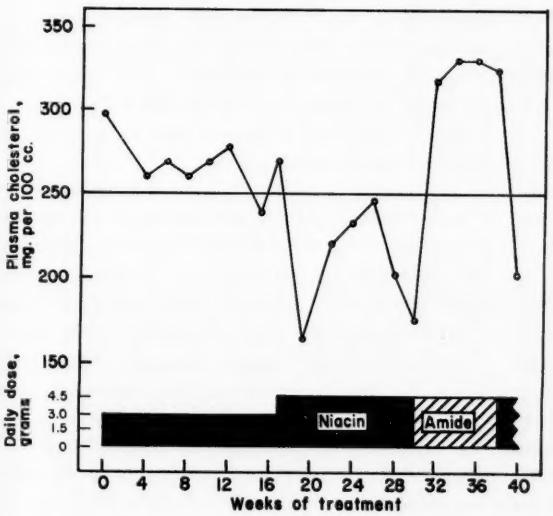


Figure 3.—Illustration of reduction of blood cholesterol levels to normal by administration of 4.5 gm. of nicotinic acid daily, return to elevated levels when nicotinamide was substituted, and reduction to normal levels when the use of nicotinic acid was resumed.

Illustrations courtesy J.A.M.A. **165**: *234-238 (September 21) 1957.*

pretreatment values within a few days or a week. Patients continue to eat their normal diets throughout the study.

Figure 2 shows the results in a typical case with good response to 3 grams daily. Figure 3 shows a good response to 4.5 grams daily. The average cholesterol levels in 44 patients in the various treatment periods are shown in Table 2. Table 3 shows the results in the lipoprotein fractions in Group 2 as well.

The flush and pruritus that occur after administration of nicotinic acid subside rapidly in the first few days or week of therapy. When flush persists past the first two weeks, it is mild and has not in any case required discontinuation of therapy. No abnormalities in hematopoietic, cardiovascular, renal, or hepatic function have been found. Because of a study showing fatty changes in the livers of rats when an excess of nicotinamide was fed in the diet,[4] we recently performed a battery of liver function tests on 19 patients and needle biopsies of the liver on 17 who had been in the study for longer than one year. No evidence of hepatic dysfunction or significant morphologic change was found.

The mechanism of action is not known, but we are studying differences between the action of nicotinic acid and nicotinamide in an effort to account for the effect of the former on cholesterol metabolism.

Table 2.
Average Serum Cholesterol Levels* Following Treatment with Nicotinic Acid and Nicotinamide

	Pts.	Pre-Rx	0-12 wks. (N.A.)	12-30 wks. (N.A.)	30-42 wks. (amide)	42-56 wks. (N.A.)
Group 1	26	325	263	259	331	277
Group 2A	9	315	278	274	342	——
2B	9	301	262	248	——	——

*mg. per 100 cc.

CLINICAL TREATMENT

To summarize our present view of treatment, we feel that — at least, in younger persons with hypercholesterolemia — some form of therapy should be employed if the patient desires it and understands that he is taking part in a clinical experiment. The results should be evaluated by several control observations before treatment and repeated tests during treatment. If the patient is obese, restriction of fat and calories in the diet might be the first method tried. If this program fails to produce normal cholesterol levels (or normal beta lipoprotein cholesterol levels, if they are determined) or if the patient is not obese, he might be induced to try a program of restriction of saturated fats with the addition of unsaturated fats, or large doses of nicotinic acid can be used without change in the diet.

I do not wish to leave the impression that treatment of hypercholesterolemia is the same as treatment of the hypercholesterolemic patient. Frequently, knowledge of the presence of hypercholesterolemia produces undue anxiety, which usually can be allayed by a discussion presenting the available information in proper perspective. When clinical manifestations of atherosclerosis or its complications are present, they should be treated on their own merits, as should such related disorders as arterial hypertension, diabetes, or hypothyroidism.

Finally, one of the great dangers facing us in this field is the possibility of widespread uncritical use of one or a combination of the available agents with the philosophy that "at least it can do no harm." This might result in over-the-counter sale of alleged preventives for atherosclerosis surpassing in magnitude the abuse of vitamin preparations that has made vitamins the greatest money-makers in the drug industry. Each of us has a part in striving to prevent such a situation.

SUMMARY

1. Because of statistical evidence incriminating hypercholesterolemia in atherogenesis, clinicians are justified in attempts to reduce elevated cholesterol levels provided both physician and patient understand they are conducting a clinical experiment, not using a method of established value for the prevention of atherosclerosis.

2. Several clinical clews are available to assist in detecting patients with hypercholesterolemia, but many cases are found by routine screening.

3. Of the numerous methods shown experi-

Table 3.
Average Beta- and Alpha$_1$-Lipoprotein Cholesterol Levels* Following Treatment with Nicotinic Acid and Nicotinamide
(Group 2—18 patients)

		Pre-Rx	0-12 wks. (N.A.)	12-30 wks. (N.A.)	30-42 wks. (amide)
Group 2A	Beta	204	159	155	228
(9 pt.)	Alpha$_1$	45	57	62	54
	B/A$_1$	5.0	2.9	2.8	4.6
Group 2B	Beta	196	152	151	——
(9 pts.)	Alpha$_1$	39	56	58	——
	B/A$_1$	7.0	3.1	3.1	——

*mg. per 100 cc.

mentally to lower serum cholesterol, those most applicable clinically are restriction of dietary fat and calories, restriction of saturated fats with the addition of unsaturated fats, and the use of large doses of nicotinic acid without dietary restriction. Other chemotherapeutic agents undoubtedly will be developed as a result of intensive programs now in progress.

4. Until a test for the presence of uncomplicated atherosclerosis becomes available, demonstration of the ultimate value of any method of reducing blood cholesterol levels must await long-term controlled studies in humans to show the comparative incidence of complications of atherosclerosis.

REFERENCES

1. Gofman, J. W., and others: Evaluation of Serum Lipoprotein and Cholesterol Measurements as Predictors of Clinical Complications of Atherosclerosis, Circulation 14:691-741 (Oct.) 1956.
2. Page, I. H.; Stare, F. J.; Corcoran, A. C.; Pollack, H.; and Wilkinson, C. F., Jr.: Atherosclerosis and the Fat Content of the Diet, Circulation 16:163-178 (Aug.) 1957.
3. Achor, R. W. P.; Berge, K. G.; Barker, N. W.; and McKenzie, R. F.: Treatment of Hypercholesterolemia with Nicotinic Acid, Circulation 17:497-504 (April) 1958.
4. Handler, P., and Dubin, I. N.: Significance of Fatty Infiltration in Development of Hepatic Cirrhosis Due to Choline Deficiency, J. Nutrition 31:141-159 (Feb.) 1946.

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Do you know your HQ (heart quotient)?

Do most people who have heart disease die suddenly?

Are there many kinds of heart disease?

Do you know what services there are in your community for people with heart disease?

A Heart Quiz containing 20 questions like these together with answers will be sent upon request. Write the Chicago Heart Association, 69 West Washington, or call Financial 6-4675.

Each year in Chicago more than 300 children die of congenital heart defects. To combat this situation, the Chicago Heart Association has initiated, with the United States Public Health Service, the biggest project ever undertaken in this field. The project is known as the Congenital Heart Institute which has the expressed co-operation of Chicago's five medical schools and the health agencies of city and state.

Parents — a reminder — it is particularly important for your child to have a physical check-up at the beginning of the school year. Actually, a heart examination is part of every complete physical examination. Now, says the Chicago Heart Association, is a good time to make a date with your family physician for that examination.

Your heart is an efficient machine, more efficient and more enduring than any devised by man. In the course of a day it pumps 10 thousand quarts of blood into 12 thousand miles of arteries. If you would prove how much energy this involves, visit the heart exhibit of the Chicago Heart Associations at the Museum of Science and Industry in Jackson Park, and work the electric hand grip in time to your normal heart beat —72 times a minute. See how tired you become. Then remember that your heart works this hard continuously from birth to death. *Do You Know Your HQ (Heart Quotient)? J. School Health Nov. 1957.*

Chemosurgery in the Treatment of Cancer: Its Scope and Indications for Its Use

HENRY A. SZUJEWSKI, M.D., CHICAGO

The problem of tumors of the skin, since the beginning of recorded medical history, has presented one of the greatest challenges to the ingenuity of the surgeon. The fundamental goal then, as now, is complete eradication of the growth. The limitations of surgery were considerable before anesthesia and blood replacement were known. The discovery of roentgen rays brought new hope for the cancer patient. For easily accessible cancers this form of therapy was thought by some to be the final answer. Advancements in anesthesia and blood replacement therapy allowed bolder surgical attacks on these accessible cancers.

Despite these advances, appreciable numbers of cancers of the skin and other accessible areas recurred following treatment. The commonest reason for these recurrences is that the original tumor was incompletely removed or destroyed. Moreover, the apparent recurrence may be a new primary tumor in the same or adjacent tissue, probably related to the same etiological factors as the previous primary tumor.

Incomplete destruction or removal of a cancer is due generally to one of two factors or a combination. First, cancers do not grow uniformly in all directions, but grow and extend in irregular, often bizarre patterns. The direction and magnitude of these extensions rarely are apparent to even the most experienced observer. The second major factor in recurrence is the unwillingness of the surgeon to sacrifice larger areas of what appear to be "normal" tissues. Especially is this true in lesions of the face where such excisions would result in difficult or impossible reconstructive surgery. Recurrences are not limited to surgical treatment alone. They are equally common following the various forms of radiations and for the same reasons.

There is evidence that the development of carcinoma is not dependent entirely upon changes in the epithelial cells. Local tissue inhibitory factors are capable of keeping malignant development in check. Removal of such an inhibitor may be the actual immediate cause of cancer[5]. This inhibitor is not a fixed property of the tissues but can be made to shift by experimental interference. It can sometimes be broken down locally by cautery, curetting, and other trauma of the tissues, in an area where there is a hyperplastic condition of the epithelium. Malignant changes can follow.

The variety of cell types and bizarre extensions of cancer of the skin probably depends upon the constancy of the inhibitor, i. e., its presence or absence and in what amounts. The degree of malignancy depends upon the capacity of the organism to produce the inhibitor. The behavior of epithelial cancer in all organs probably mirrors the behavior of cancer of the skin.

Whatever the exciting agent may be, the connective tissue apparently responds to the insult in a manner compatible with organismal control. It may be that this varied response on the part of the epithelial mesenchyme and endodermal structures is responsible for the numerous cell types and varied growth patterns observed in cancer. These different types of cancer re-

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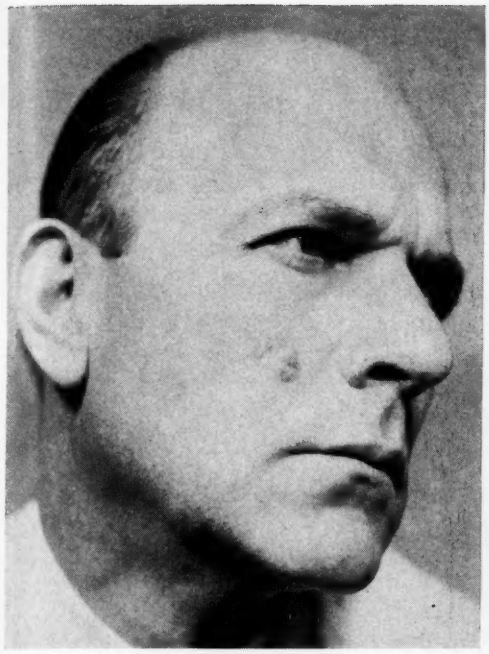


Figure 1. Case G.P. Preoperative photograph shows area of neoplasia, right cheek.

Figure 2. Photomicrograph of chemosurgical biopsy specimen obtained at first excision. Frozen technique used.

spond differently to treatment. Uniformity of response can be achieved only by a form of treatment that encompasses all types of growths, regardless of morphology. Chemosurgery is the only presently known form of treatment answering this need.

This method can be employed for skin cancers no matter what the cell type of the tumor may be. Repeated performance of the chemosurgical technique on cancer of the nose[1], ear[2], eyelids[3], and face[4] confirms this principle. The high percentage of cancer-free years enjoyed by patients who have been treated for recurrent cancer supports the effectiveness and thoroughness of the chemosurgical technique.

A case is presented to show the actual application of the technique in the treatment of a specific case:

Mr. G. P. (Figure 1), aged 47, complained of "something growing" on the right cheek. The growth was first noticed about five years ago when a pulling sensation was experienced. About a year ago his friends began to comment on the "thing" on his face. No treatments had been administered at area on the right side of the face, with slight bluish discoloration. The edge seemed glistening. There was no bleeding. The rest of the physical examination was negative. The impression before biopsy was that the patient had a basal cell carcinoma that seemed quite large to palpation.

First Day: Dichloracetic acid was applied superficially over the visible nodule. Whitening of the nodule and adjacent area indicated adequate penetration of the keratin and facilitated the passage of zinc chloride. Zinc chloride fixative was applied superficially over the area to the depth of about 2 mm. A dressing was applied carefully to exclude air.

Second Day: The dressing was removed. A layer of tissue from the treated area was excised, the incisions being made within the killed tumor tissue so that there was no pain on cutting and no bleeding. The first specimen thus removed was saved for microscopic examination. The microscopic diagnosis was basal cell carcinoma, invasive in character (Figure 2). Fixing agent was reapplied, and the treated area covered with a dressing.

Third Day: Dressing was removed and another layer of tissue dissected free. Microscopic examinations were made. The excised layer was divided into separate specimens and their edges marked for orientation with mercurochrome (solid lines), ordinary bluing (dotted lines), and black indigo ink

Figure 3. Diagram showing how tissue specimens are excised and marked for orientation.

Figure 4. Final layer has separated by itself and a granulating self-healing area now remains.

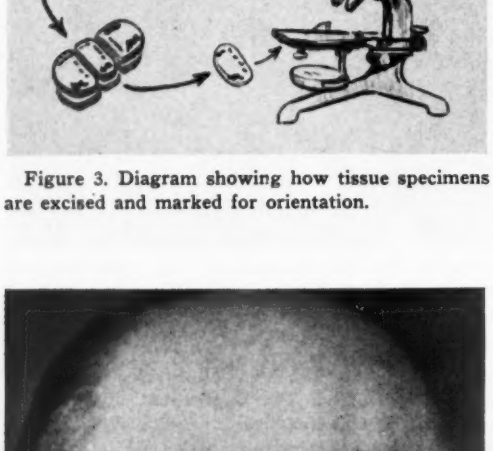


Figure 5. Wound now healed in less than three weeks. Minimal defect with small scar that will become less conspicuous with time.

(jagged lines) (Figure 3). Frozen sections were cut through the under surface of each specimen. Microscopic examinations revealed cancer in all specimens, as indicated on map. The fixing agent was applied once more to the corresponding areas of the lesion.

Fourth Day: Specimens were removed from the treated areas, and a few areas were found positive for cancer. These were treated again.

Fifth Day: Specimens were removed and re-examined. No evidence of cancer was found. Vaseline® dressing was applied.

Ninth Day: The final layer of fixed tissue separated itself and was removed (Figure 4). Vaseline dressing was applied. Within three weeks the lesion was healed (Figure 5).

The main advantage of this technique is that the thorough rigid microscopic control allows all tumor tissue to be removed while sacrificing minimal amounts of normal tissue. There are other important advantages. One of these arises from the gentle handling of the tumor and its bed which are inherent in this technique. There is no sudden disturbance of the entire mass of the tumor, as in curettage, electrocauterization, or surgical manipulation of a tumor. There is no interference with the balance between host resistance (connective tissue) and the tumor itself. Both are chemically fixed, thus bringing about

a state of complete stability of the tissues involved in the tumor area. Another advantage of chemosurgical method is the early availability of adequate material for biopsy. There is no substitute for a good biopsy. In chemosurgery there is no interruption of the treatment as may be the case in waiting for fixing, sectioning, and staining paraffin sections. Specimens are made immediately for microscopic examination and may be interpreted several minutes after the first excision. The microscopic examination of frozen sections extends tissue examination of epithelial and connective tissue elements because chemosurgical fixation of tissue does not greatly alter the noncellular or the cellular structure[6]. The dehydration and shrinkage involved in paraffin embedding tends to alter the noncellular tissues to such an extent that fibrin may be almost indistinguishable from fibrous connective tissue.

The great anatomical variation that exists in skin cancers is no contraindication for the use of this technique, whereas it may be with radiation therapy or surgery. So great is the variety of architecture in skin cancers that they should be dealt with separately in the organs in which they occur. Therapy should not be matter of speculation, but ought to be precise in that identification of the tumor and peritumor tissue should be thorough.

Diffuse infiltrating tumors, the cells of which are separated by dense connective tissue, are less likely to respond to X-ray or gamma rays than superficial solid type tumor without much connective tissue surrounding it. The morphologic characteristics of a tumor indicate the varying degree of organismal control more often than is suspected.

It is logical to assume that therapy to a tumor often interferes with organismal control so that areas of neoplasia actually become more active and virulent in their progressive destruction of the host. Excessive radiation, radical surgery, or any other therapeutic force capable of unbalancing the tumor host stability, often hastens the death of the patient rather than enhancing the control of the neoplastic process. It is in this class of skin tumors where the highest percentage of recurrence takes place[3]. The center of the tumor frequently is well condensed with scar tissue, giving the appearance of healing. This appearance is often found following X-ray ther-

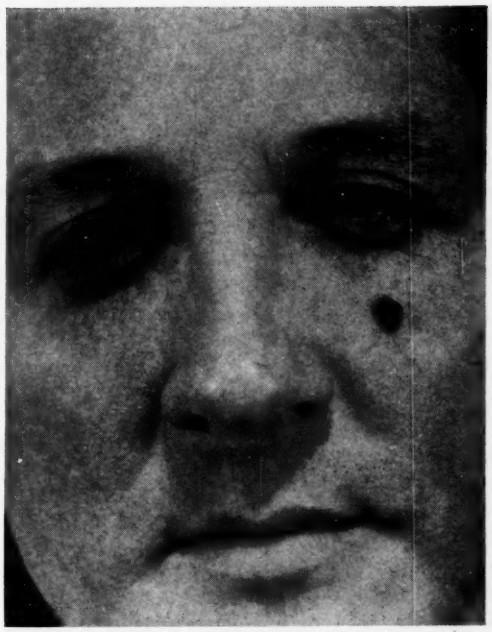


Figure 6. The halo-like area to the right of the cancerous nodule is scar tissue that appears to be tumor-free, but actually tumor is present between the fibrous connective tissue and even under it in the subcutaneous tissue.

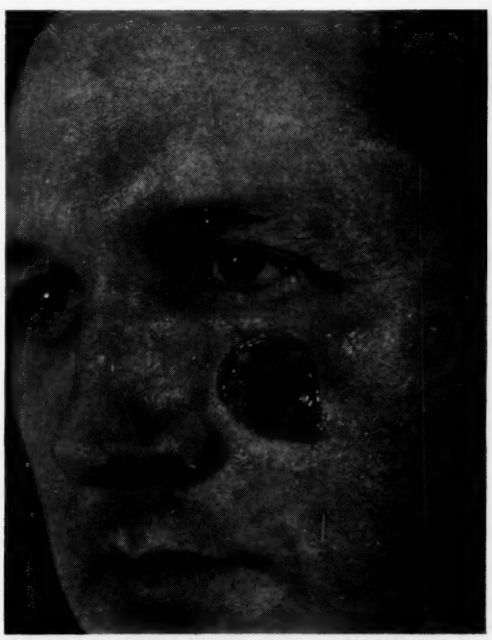


Figure 7. Entire area of scar harboring tumor cells now removed along with cancerous nodule which manifested itself at periphery of scar, where tissues were less dense.

apy or desiccation (Figure 6). Actually, the scar tissue harbors cancer that finally reappears when less dense tissue is invaded at the perimeter of the lesion (Figure 7).

The time of recurrence depends, it seems, on the density and extent of the scar tissue, and not on the viability of the malignant cells themselves. If cancer cells are caught within scar tissue and have to make their way through it, the time interval between remission and reappearance is longer. If cancer cells are only partially limited by scar and gain access to soft pliable tissues, recurrence appears more frequently and sooner.

The location of the skin tumor becomes of great importance when therapy is selected. A tumor near or on the nose, on the concha of the ear, margin of the eyelid, or tip of the nose becomes a technical problem when either the surgeon or the radiologist considers therapy. Because of the tendency to spare tissue and because of the hazard of radiation reaction on cartilage, tumors in these areas frequently are incompletely removed or treated. The architecture of the epithelial structures and the cementing tissues often is totally ignored. In addition, reactions in connective tissue overlying cartilage and bone differ, so far as ability to respond to varying degrees of injury as compared to connective tissue reaction in neck, abdomen, thigh, and buttocks.

The result is that there are frequent recurrences at or near these sites, and injuries to normal tissue result far more often than necessary. Skin folds or cartilage offer no difficulty when the chemosurgical technique is used. The resulting scars are minimal and grotesque defects, practically nonexistent. The surgery has been conservative in that tissue containing malignant cells is removed, and at the same time radical, since complete removal of tumor has been accomplished.

Experience seems to point to the following indications for use of the chemosurgical technique:

1. Primary accessible malignant tumors of the skin, except malignant melanoma which should be radically excised. Although Mohs[7] reports successful treatment of melanoma by chemosurgery, he emphasizes that it is necessary to remove an additional zone of tissue after a melanoma-free level has been reached because of the danger of satellite deposits in the surrounding lymphatics. If satellite lesions are present around a melanoma, it is quite certain that more distant metastases are about to occur or have already occurred. Radical excisional biopsy for a melanoma, in most cases, may cure a patient. Node dissection is not necessary when satellite lesions or suspicious nodes are not present. When metastases are present at the time of discovery and diagnosis, en bloc dissection and radical surgery certainly should be attempted. Since malignant melanoma does not present iceberg growth pattern but a centrifugal growth, radical surgery seems more logical and the attack becomes swifter in time than in the chemosurgical attack. Again, the melanomas vary widely in their relation to physiological processes rather than anatomical features.

2. After a single recurrence, chemosurgery should be instituted at once. A tumor that has been cauterized previously by an electric unit, radiated, or surgically treated and recurs, follows an extremely bizarre growth pattern after such treatment. The growth kinetics are altered. The spread of the tumor is no longer totally affected by organismal control but only partially so. Such a tumor is best treated by total identification under the microscope as well as totally removed at the same time. In some cases, where there is recurrence despite chemosurgical removal, subsequent changes in the tissue of a given area may or may not be related to the original cancer and cannot be assigned as failure of the method. This is especially noticeable in only a very limited number of cases that have been subjected to multiple methods of therapy, especially heavy radiation. There is the possibility that isolated outlying foci of cancer might result either from surgical or radiation treatment. Since these foci are disconnected from the main mass of cancer, they might be missed despite the microscopic control afforded by the chemosurgical technique. This is true also in cases where cancer is associated with such diseases as lupus erythematosus or psoriasis.

3. In external cancers of the nose and of the concha of the ear, minimal destruction of these organs is obtained, yet total removal of tumor is accomplished. (Figures 8, 9, 10, 11).

4. In cancer of the margin of the eyelids or on the eyelids themselves, the eye is not injured by the chemosurgical technique and maximal

Figure 8. Preoperative photograph showing surgical scar with reappearing tumor. Chemosurgery now used.

Figure 9. Photograph shows how extensive and deep the cancer actually invaded the nasal tissues.

Figures 10 and 11. Patient completely rehabilitated. Nasolabial full thickness pedicle graft. Reconstruction of Dr. Wayne B. Slaughter. Patient well now for five years plus.

preservation of noncancerous eyelid tissue is achieved. If the inner canthus of the eye or the tear duct itself is infiltrated with tumor, these areas are easily made free of tumor with the chemosurgical method.

5. In cancers that arise in previously radiated skin, it is obvious that more radiation is contraindicated. Since multiple foci of cancer have their greatest incidence in overradiated skin, such skin may be removed and replaced by split thickness or pedicle graft. Such procedures are quite radical and cosmetic results are far from satisfactory. Multiple foci of cancer in such skin can be treated chemosurgically and the scars are less conspicuous. The rest of the skin of the face is benefited also because during the treatment there is an increase in the capillary blood flow due to the vasodilatation, which is part of the inflammatory reaction accompanying the treatment. Medical management following the removal of cancer in radiated skin is necessary and mandatory if further development of cancer is to be kept at a minimum or inhibited completely. A description of such management will be presented in a future report.

6. Cancers of the scalp, especially those having their origin in hair follicles can be efficiently eradicated with little loss of hair and with no injury to underlying bone. Skin graft and other reconstructive surgery rarely is needed following chemosurgery, except in very extensive lesions.

7. Cancers of the lip can easily be eradicated by chemosurgery. Large and matured cancers of the lip should be removed by radical surgery. When the classical V resection is employed, it is best to include most of the mucous membrane of the lip, especially when it is even slightly thickened, pale, or fissured. Cancers at the angle of the lip or at the mucocutaneous junction are best treated chemosurgically, especially if they are of the basal cell variety or spinobaso type. So-called leucoplakias that are fixed to the mucous membrane with fissure formation, usually are already early cancer. They should be removed in all cases and the base of the lesion thoroughly examined with the microscope until all evidence of neoplasia is removed.

8. Cancer of the penis can be treated adequately by chemosurgery if the tumor is confined to the glans penis.

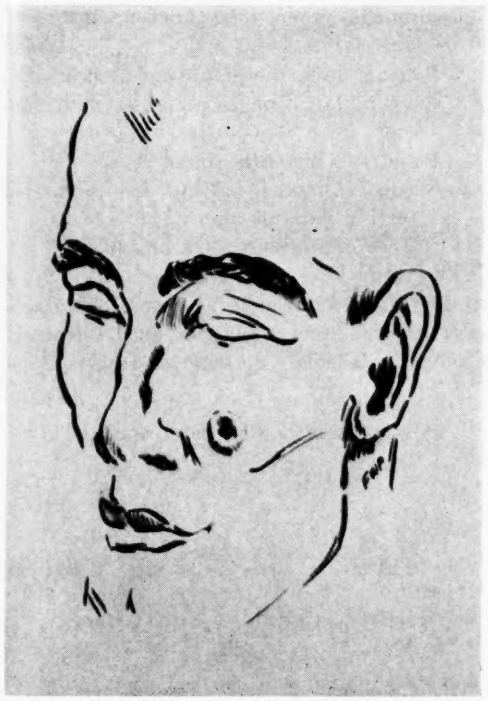
9. Cancers that have their origin in chronic skin conditions, especially those that have had long previous treatment, are best treated chemosurgically. This includes lupus erythematosus, acrodermatitis, psoriasis, extensive burns with keloid formation, and xeroderma pigmentosa. They are closely allied to problems in which multiple cancers arise in radiated skin. In this category only are found the most difficult cases that do not respond completely to any form of treatment. It is felt that these cases are not cancer problems alone, but that other debilitating diseases are contributing to the cancer diathesis.

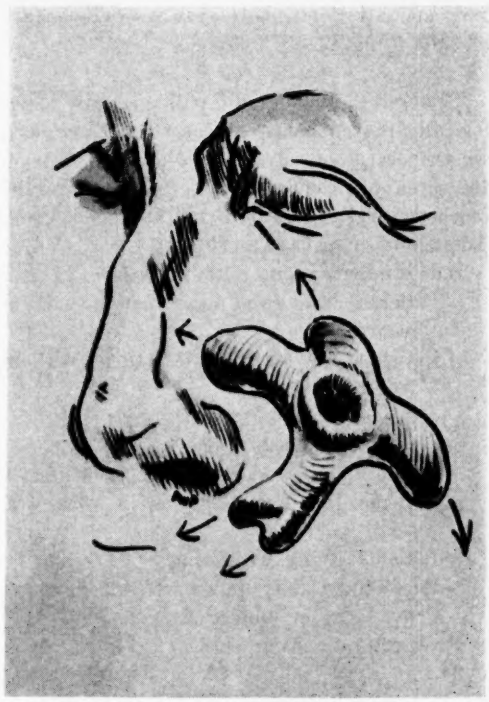
10. The chemosurgical technique also is useful in the treatment of other neoplastic and preneoplastic lesions of the face and skin, such as hemangiomas, nevi, keratoses, papillomas, xanthomas, and granulomas. Results in the treatment of these lesions are most satisfactory.

11. Chemosurgery also is indicated for many cancers in cavities accessible through normal, artificial, or pathologic openings by using modifications of the technique. This includes neoplasms of the lips, oral cavity, salivary glands, nasal cavities, accessory sinuses, larynx, vulva, vagina, penis, and anus. Local recurrences following mastectomy often can be treated successfully by this method. This list of indications is not necessarily complete. Other uses of the method are constantly being enlarged.

In all cases where lymphatic spread is detected, subsequent radical neck or regional node dissections must be performed. The chemosurgical technique for treating cancer is based on a thorough knowledge of surgical principles, staining of tissues, microscopic interpretation of tissue specimens, pathology in all its forms, and tissue reaction and repair. The success of the chemosurgical method depends on scrupulous attention to details. The technique is unexcelled for its reliability and accuracy, when properly applied. Its place in the treatment of accessible cancer is unique because of the microscopic guidance it offers.

Microscopic control is necessary because of the strong tendency for epitheliomas to exhibit irregular and often unsuspected outgrowths from the main cancerous mass and because of the great diversity in morphology. These bizarre outgrowths are more extensive in advanced and in recurrent cancers, but not infrequently they are observed in early lesions or untreated areas





Figures 12 and 13. Diagram demonstrating that epitheliomas of the skin exhibit irregular and often unsuspected outgrowth from the main cancerous mass before or, more often, after ineffective therapy.

as well. (Figures 12 and 13). The use of the chemosurgical method is most helpful in the treatment of accessible cancer in the aged; the method entails practically no mortality. Although many cases were in an advanced age group, no death occurred during treatment.

The only disadvantage of the method is that specialized training and experience are required for the attainment of best results.

The following charts show the end results of the chemosurgical treatment of basal cell carcinoma and squamous cell carcinoma of the skin of the face after a period of five years and over.

CHART 1.

This series includes all patients with histologically proved basal cell carcinoma, both early and advanced, not previously treated, who were treated chemosurgically during the five year period ending July, 1957.

Total number of cases: 38

Indeterminate group, total number: 2

Patients dead from other causes without recurrence: 1

Patients lost from observation without recurrence: 1

Determinate group, total number 36

Unsuccessful results: 0

Patients dead, cancer present at death: 0

Patients lost from observation with cancer: 0

Patients living with cancer: 0

Successful results, patients free from cancer five years or more: 36

Five year rate of cure: 100%

Basal Cell Carcinoma

		five year cure
Nose	10	100%
Face (cheeks)	19	100%
Forehead	2	100%
Concha ear	2	100%
Skin of back	2	100%
Mucocutaneous junction	1	100%

These cases were all of the infiltrating type. The size of the lesion varied from 1 to 10 cm. The first treatment in all cases was chemosur-

gery and all were consecutive cases. They are all still being closely followed.

CHART 2.

This series includes all patients with histologically proved recurrent basal cell carcinoma, whether early or advanced, who were seen in the seven years ending July, 1957. The results of chemosurgical treatment are tabulated below.

Total number of cases: 20
- Indeterminate group, total number: 1
  - Patients dead from other causes without recurrence: 1
  - Patients lost from observations without recurrence: 0
- Determinate group, total number: 19
  - Unsuccessful results: 1
  - Patients dead, cancer present at death: 0
  - Patients lost from observation with cancer: 0
  - Patients living with cancer: 1
  - Successful results, patients free from cancer five years or more: 18
- Five year rate of cure (18-19) x 100% equals 94.7%

The adverse effects of previous treatment on prognosis are indicated by the rate of cure, 94.7% (Mohs 95%) for these patients with recurrent lesions, as compared to the rate of 100% for those who had not received previous treatment.

The fact that 94.7% of all recurrent lesions responded to chemosurgical treatment indicates the effectiveness of the method. Most of these cases were far advanced and many had been operated or heavily irradiated or both. In this series all cases are still alive except the one in the indeterminate group, and all are still being followed.

CHART 3.

This series includes all patients with histologically proved squamous cell carcinoma, whether early or advanced, primary or recurrent, with or without metastases, who were treated chemosurgically during a seven year period ending July, 1957.

Total number of cases: 11
- Indeterminate group, total number: 0
  - Patients dead from other causes without recurrence: 1
  - Patients lost from observation without recurrence: 0
- Determinate group, total number: 11
  - Unsuccessful results: 0
  - Patients dead, cancer present at death: 0
  - Patients lost from observation with cancer: 0
  - Patients living with cancer, 0
  - Successful results, patients free from cancer five years or more: 11
- Five year rate of cure: 11 or 100%

The method for reporting results is the one used by Dr. F. Mohs in his new book, "Chemosurgery and Cancer, Gangrene and Infection," Charles C. Thomas, Springfield, Illinois, 1956.

Squamous Cell Carcinoma

		five year survival
Nose	5	100%
Face (cheek)	2	100%
Upper lip	2	100%
Skin (neck)	2	100%

The grading of these cancers (Broder's) ranged from grade I and grade IV.

SUMMARY

A discussion of the underlying principles affecting the growth of cancer of the skin is presented as well as the necessity for a thorough microscopic eradication of the skin cancer. The scope and indications for the use of the chemosurgical method are described. Experience with the method gives an unequaled appreciation of its effectiveness. By seeing the method in use, its thorough and complete attack on cancer can be fully appreciated. It is a logical method since it does not interfere in any way with effort by the host to combat the tumor. Chemical fixation of a tumor in situ and its removal results in a truly dramatic healing response. Detection and treatment of early cancer by this method is so easily achieved it is truly one of the most effective methods in the control of cancer.

55 E. Washington St.

REFERENCES

1. Mohs, F. E.: Chemosurgical Treatment of Cancer of the Nose: A Microscopically Controlled Method, Arch. Surg 53:327-344, 1946.
4. Mohs, F. E.: Chemosurgical Treatment of Cancer of the Ear: A Microscopically Controlled Method of Excision, Surgery 21:605-622, 1947.
3. Mohs, F. E.: Chemosurgical Treatment of Cancer of the

Eye-lid: A Microscopically Controlled Method of Excision, Arch. Ophth. 39:43 (Jan.) 1948.
4. Mohs, F. E.: Chemosurgical Treatment of Cancer of the Face: A Microscopically Controlled Method of Excision, Arch. Dermat. & Syph. 56:143-156, 1947.
5. Cramer, W.: On Experimental Carcinogenesis. The Local Resistance of the Skin to the Development of Malignancy. Brit. J. Exper. Path. 335:10, 1929.
6. Duguid, J. B.: Thrombosis as a Factor in the Pathogenesis of Aortic Atherosclerosis, J. Path. & Bact. 60:57, 1948.
7. Mohs, F. E.: Chemosurgical Treatment of Melanomas: A Microscopically Controlled Method of Excision, Arch. Dermat. & Syph. 62: 269-279 (August) 1950.
8. Szujewski, H. A.: Microscopic Guidance in the Treatment of Skin Cancer, Plast. and Reconstruct. Surg. 5:6 (June) 1950.

Calcific Aortic Stenosis: Diagnostic Considerations

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BECAUSE of recent advances in the surgical treatment of valvular heart disease, it is imperative that all cardiac murmurs be evaluated correctly. This paper is being presented to point out the difficulties in the diagnosis of aortic stenosis and to offer suggestions to aid in its diagnosis.

Aortic stenosis as a cause of cardiac disease is encountered frequently and is becoming more and more important as the age of our population increases. In this paper we are interested in the calcific form; the question of whether or not it is related to rheumatic fever is not within its scope. In the past few years there have been important advances in the surgery of stenotic lesions of the aortic valve. Surgery is not indicated for all, but every attempt to diagnose the condition should be made so that thought can be given to surgical therapy. With the advances in technique and anesthesia, age is no longer the deterrent to surgery it once was. Witness the recent elective gall bladder surgery on Herbert Hoover when he was 83 years old.

Formerly, various criteria for diagnosing aortic stenosis were taught in our medical schools, hospitals, and textbooks. Now we are finding that rigidity in the application of these criteria may cause the diagnosis to be missed. Because of two recent instances of incorrect diagnosis the records of Rockford Memorial Hospital were reviewed. Of 23 patients with an autopsy diagnosis of severe to moderate aortic stenosis, the diagnosis was missed clinically in 11 patients. There are many articles in the literature on aortic stenosis but, because of the large number of patients and the thoroughness of the studies, two were selected for discussion. The first was published in 1948[1] and the second in 1954[2]. In the first series the correct diagnosis was made in only 41 per cent* of the 107 patients. In the second study, the correct diagnosis was made in only 50 out of 100 patients. Many of these patients could have been helped by surgery. This article is being written in an attempt to increase the accuracy of the diagnosis so that more patients can be offered help.

Classically, the patient with aortic stenosis presents with a loud rough aortic systolic murmur, an aortic systolic thrill, an absent or faint second aortic heart sound, a characteristic small delayed pulse, and a low pulse pressure. In our small series at Rockford Memorial Hospital, and in the two previously mentioned larger series, the above complete findings were present in a definite minority. Reliance on these signs was more of a hindrance than a help in diagnosis

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*Note: The article stated that the correct diagnosis was made in only 26 per cent, but 15 per cent had a diagnosis of combined valvular disease.

because of their rareness. When present they are significant because they lend confirmation. In these series the "typical" level of the blood pressure and the type of pulse were so unusual that they should be disregarded completely as diagnostic signs. The aortic second sound was decreased or absent, or less than the pulmonic second sound in only 50 per cent of the patients. Even with severe aortic stenosis, with just a small blood flow, the aortic second sound may be loud and booming. In some of these patients, the aortic second sound may be transmitted from the pulmonic area. It is suggested that the aortic second sound may be evaluated more completely by listening for it in the first right intercostal space near the sternum. Even the incidence of a basal systolic thrill was low, being found in only 21 per cent[1] and 45 per cent[2] of the cases. In the group with the larger percentage, the thrill was searched for with the patient leaning forward and the breath held in deep expiration.

Of the classical criteria the loud rough systolic aortic murmur is the most reliable; but even this is not too reliable. In about 10 per cent of the patients no murmur will be heard because of loud respirations or pulmonary emphysema. In the remainder only about 70 per cent will have a rough aortic systolic murmur. So, even when there is every opportunity to hear the murmur, it will only be heard in about 70 per cent of the patients.

How is the diagnosis made in the remaining patients? This group does have a finding that is of much value. Those without a basilar systolic murmur will have a characteristic apical murmur 70 per cent of the time. The murmur frequently is described as harsh, squeaking, or musical. When this systolic murmur is heard at the apex, careful search should be made for a basilar systolic murmur which, when found, often is soft, yet harsh.

From the above we can see that the most reliable of the criteria for the diagnosis of aortic stenosis is a harsh or musical systolic murmur heard either at the base or apex of the heart. A harsh systolic mumur also is heard with numerous other conditions which should be considered in the differential diagnosis. Probably the most common cause of a harsh aortic systolic murmur—other than aortic stenosis—is dilatation of the aorta due to arteriosclerosis. Here the murmur is less harsh, the aortic dilatation is seen radiographically, and there is no calcification of the aortic valves.

Another condition commonly mistaken for aortic stenosis is mitral insufficiency. The murmur of mitral insufficiency may be transmitted to the aortic area and into the neck. But if it is transmitted this widely and loudly, it is easily heard over the left posterior lung base, and very indistinctly, over the right anterior chest; whereas the murmur of aortic stenosis (even if loudest at the apex) is not heard at the left lung base. Also, uncomplicated mitral insufficiency is rare after age 50 and does not show aortic valve calcification.

In the age group in which aortic stenosis is most commonly found, another condition frequently is present: calcified mitral annulus. Here again we have a harsh systolic murmur usually heard best at the apex. The transmission of the murmur usually follows that of mitral insufficiency, but the most important differential point is the timing of the murmur. It does not immediately follow the first heart sound and usually is described as mid-systolic. It begins in the middle of systole and builds up until it ends with the second heart sound or soon after. The murmur of aortic stenosis begins soon after the first heart sound, builds up into a crescendo, followed by a decrescendo, giving rise to a characteristic diamond-shaped murmur. In calcification of the mitral annulus the calcification usually is seen fluoroscopically as a dense, ringlike area of calcification with little pulsation.

Another condition sometimes diagnosed instead of aortic stenosis is aortic regurgitation. This is understandable because of the frequent coexistence of an aortic diastolic murmur and high pulse pressure even with severe aortic stenosis. Since the two lesions may coexist, accurate clinical judgment is needed. Frequently the diagnosis as to the important lesion can be made only by cardiac catheterization with measurement of the pressure gradient between the left ventricle and the aorta. Congenital cardiac lesions, thoracic tumors, and aortic aneurysms usually can be easily differentiated by the age of the patient and by X-ray examination of the chest.

The positive aspects of diagnosis will be emphasized now. If we make the same effort to diagnose aortic stenosis as we do to diagnose mitral stenosis, I believe our diagnostic accuracy will

be almost as good. Our efforts will not receive as great a reward as with mitral stenosis since many patients with aortic stenosis are aged and have other complicating diseases; but, even if we can help one in 10 our efforts will be rewarded.

On examining the patient with suspected aortic stenosis (and everyone with heart disease should be a suspect) we should look for the "classical" signs of aortic stenosis such as a systolic thrill and low pulse pressure. But we must not be disappointed not to find them. We ought to look mainly for a harsh systolic murmur, loudest in the aortic area, and transmitted to the neck and right shoulder. At times the murmur will be loudest at the apex, but it is not transmitted to the left lung bace posteriorly. The timing and tonal characteristics of the murmur are important whether heard at the apex or over the base of the heart. The murmur begin at, or soon after, the first heart sound, builds up in crescendo fashion, and abates as a decrescendo giving rise to a diamond-shaped murmur. The murmur is harsh, or squeaking, or high-pitched, or musical. When thought to be heard exclusively at the apex, careful examination of the patient at the base of the heart frequently will result in the hearing of a soft but harsh aortic systolic murmur.

If aortic stenosis is suspected the patient should be fluoroscoped for calcification of the aortic valve[3,4,5]. In the right anterior oblique position, the aortic valve lies somewhat above and anterior to the mitral valve. In the left anterior oblique position, the aortic valve lies in the middle third of the cardiac shadow while the mitral valve is in the posterior third. The calcific valves appear as dense nodular pulsating shadows that appear to dance in a jerky fashion downward and apical in systole, and upward and toward the midline in diastole.

If aortic stenosis is suspected, and surgery to the valve is being considered, cardiac catheterization should be undertaken to ascertain the differential pressure between the left ventricle and aorta. This is done mainly to confirm the diagnosis in doubtful cases, but should be done also in patients with a diagnosis of aortic stenosis in whom surgery is contemplated. This is true since some patients with aortic stenosis will have no functional disability from it and another cause will be found for the cardiac disability.

SUMMARY

By reliance on the "classifical" findings of aortic stenosis the diagnosis will be missed about 50 per cent of the time.

The diagnosis of aortic stenosis can best be made by paying close attention to a rough basilar or apical systolic murmur: its timing, tonal characteristics, and radiation to the neck and right shoulder.

Confirmation of the diagnosis frequently can be made through cardiac fluoroscopy, looking for calcification of the aortic valve.

In doubtful cases and those in whom surgery is contemplated, cardiac catheterization should be carried out.

REFERENCES

1. Kumpe, C.W. and Bean, W.B.: Aortic Stenosis: Study of Clinical and Pathologic Aspects of 107 Proved Cases, Medicine 27:139-185, 1948.
2. Bergeron, J.; Abelmann, W.H.; Vazquez-Milan, H.; and Ellis, L.B.: Aortic Stenosis: Clinical Manifestations and Cause of Disease; Review of 100 Proved Cases, A.M.A. Arch. Int. Med. 94:911-924, 1954.
3. Sosman, Merril C.: Technique for Locating and Identifying Pericardial and Entrocardiac Calcification, Am. J. Roentgenol. 50:461-468, 1943.
4. Parkinson, J.: Radiology of Rheumatic Heart Disease (Carey Coombs Memorial Lecture), Lancet 1:896-902, 1949.
5. Davies, C.E. and Steiner, R.E.: Calcified Aortic Valve; Clinical and Radiological Factors, Brit. Heart J. 11:126-136, 1949.

The Importance of Dietary Sodium in the Etiology of Essential Hypertension

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Is excessive dietary sodium of *primary* etiologic importance in the development of essential hypertension, is it only a *secondary* permissive factor, or is it of no consequence?

Clinical Evidence For Its Importance

From the clinical standpoint, the correlation between the ingestion of a diet high in sodium chloride and the presence of essential hypertension has been extensively studied by Dahl.[1] This investigator presents evidence supporting the hypothesis that an elevated sodium intake is the primary etiologic factor in the development of essential hypertension. In a classification based on the individual's stated use of salt, he found that those on a low intake had significantly less hypertension than did those on a high salt intake. Dahl considers the eight to 10 grams of salt commonly ingested daily in this country excessive and suggests that persons who do not have a history of hypertension in the family may take up to five grams a day. Those with a family history of high blood pressure should limit the intake to less than one gram a day, to decrease the chance of developing hypertension. Other investigators who carried out similar surveys on various groups as to the amount of salt ingested and the level of the blood pressure substantiate Dahl's findings.[2]

Further clinical evidence of the importance of the level of sodium in the diet is provided by the fact that restriction of sodium intake in patients with essential hypertension results in alleviation of hypertension in some. That all such patients do not respond to this form of treatment may be due to the ability of the kidneys to reduce the output of sodium to less than the dietary intake. Chlorothiazide, a potent oral natriuretic agent, has provided a means for overcoming this renal conservation of sodium. The antihypertensive effect of this drug, which is particularly evident when it is used in conjunction with other forms of treatment, also emphasizes the benefit derived from eliminating sodium from the body.

Experimental Evidence For Its Importance

The importance of excessive sodium ingestion in contributing to elevation of blood pressure is established in some forms of experimental hypertension, such as those involving partial ablation of the adrenal glands or the administration of salt-retaining adrenal steroids. There also are several studies showing that sodium chloride alone, when given in large doses, causes hypertension in some animals. The most impressive experimental demonstration of the relationship between salt intake and hypertension, and one which provides in part the laboratory counterpart of Dahl's clinical study, is that carried out by Meneely.[3] He has shown that groups of rats fed diets containing various quantities of sodium chloride, ranging from 2.8 to 9.8 per cent, after several months, have blood pressure levels that are progressively elevated in direct relationship to the amount of salt ingested.

Just as is the case clinically, the converse of an excessive sodium intake causing hypertension is seen in the field of experimental hypertension. In some hypertensive animals, rigid sodium restriction or the removal of sodium from the body will lower blood pressure.

Clinical Evidence Against Its Importance

Not all surveys have demonstrated a correlation between the stated ingestion of salt and the blood pressure level. One study of a group of 3,000 individuals showed no relationship.[4] Some have felt that the selection of special groups for such surveys leads to bias of the data.

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While the Nutrition Committee of the Chicago Heart Association is sponsoring this article, the opinions expressed are those of the authors and do not necessarily represent the official view of that committee.

Doubt also has been expressed as to the reliability of the dietary history in respect to the exact amount of salt ingested. Studies of 24-hour urine sodium excretions of subjects on self-selected diets have shown extreme variability in the amount excreted daily.

Other clinical observations likewise minimize the primary role of sodium in the etiology of essential hypertension. Only one-fourth or one-third of all hypertensive patients treated by a low sodium diet show a fall in blood pressure. Furthermore, such subjects after a period of time on sodium restriction, may show no correlation between any change in blood pressure and the change in total exchangeable sodium.

Experimental Evidence Against Its Importance

At the experimental level it requires gross overfeeding of salt to produce hypertension. The amount necessary is many times that which humans would ingest ordinarily. Even with such large quantities, the hypertension that results occurs only inconstantly and usually with evidence of concomitant renal damage. The latter may be an important etiologic factor.

Further doubt concerning the primary role of sodium in the production of experimental hypertension is raised by the following facts: 1) The administration of the various adrenal cortical hormones does not result in hypertension in proportion to the sodium retaining abilities of these substances. 2) Potassium, when added to the diet, minimizes the effect of excessive sodium. 3) In the complete absence of potassium, additional sodium in the diet acts as a hypotensive agent.

A Rationalization Of These Differences

The data do not provide a definite answer to the question of the exact etiologic importance of excessive dietary sodium in the development of essential hypertension. However, there is overwhelming evidence that the role of sodium is extremely important. The arterial wall of the hypertensive man or animal contains more than the normal content of sodium.[5] A change in the ratio of sodium within arterial smooth muscle cells to that outside of the cells, results in an alteration in both the muscle tone and its responsiveness to vasopressor agents. Similarly, the ratio of the potassium concentration inside to that outside of these cells may be of equal if not greater importance. The proper distribution ratios of these cations are crucial for the optimal functioning of cells.

On the basis of all this information, it may be stated unequivocally that sodium plays a prominent role as a *permissive* factor in the development and persistence of hypertension. However, present knowledge strongly suggests that excessive dietary sodium is not of primary *etiologic* importance in the development of essential hypertension.

References

1. Dahl, L. K.: Salt Intake and Salt Need, New England J. Med. 258:1152-1157 and 1205-1208 (June 5 and 12) 1958.
2. Rodbard, S.: Sodium and Hypertension, Hypertension 6: 7-27 (Nov.) Proceedings of the Council for High Blood Pressure Research, 1957, published by American Heart Association, New York, 1958.
3. Meneely, G. R., and Ball, C. O. T.: Chronic Sodium Chloride Toxicity and the Protective Effect of Potassium Chloride, Hypertension 6:65-87 (Nov.) Proceedings of the Council for High Blood Pressure Research, 1957, published by American Heart Association, New York, 1958.
4. Meneely, G. R.: in Hypertension, 6:24 (Nov.) Proceedings of the Council for High Blood Pressure Research, 1957, published by American Heart Association, New York, 1958.
5. Tobian, L., and Binion, J.: Tissue Cations and Water in Arterial Hypertension, Circulation 5:754-758, 1952.

CASE REPORTS

Spontaneous Hemorrhage into the Rectus Muscle

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SPONTANEOUS hemorrhage into the rectus abdominis sheath is a clinical entity seldom considered when the physician is faced with the problem of an acute surgical abdomen. When such hemorrhage occurs, it is experienced by the patient as localized pain in the region of the rupture in the epigastric artery, which may occur anywhere along the length of the rectus abdominis muscle. Usually a mass is not palpable until the volume of extravasated blood is so large as to perfuse the adjacent muscle with blood. By the time this mass has developed, extravasation of blood has progressed by dissection down to the level of the semilunar fold of Douglas, thereby inaugurating peritoneal irritation through the thin transversalis fascia and peritoneum. If dissection does not extend to the level of the semilunar fold or beyond this area inferiorly, it is not likely to be accompanied by peritoneal irritation and is not likely to mimic clinically an acute surgical abdomen. Such cases probably go unrecognized by the clinician.

Anatomically, this area is subject to considerable stretching and contraction of the rectus muscle as well as sudden acute strains as in coughing and sneezing. This may lead to tears in the muscle fibers or their nutrient vessels and the formation of a hemorrhaging pool of blood which must follow the confines of the rectus sheath.

The etiology of this condition has been classified as traumatic and nontraumatic. Traumatic causes include those resulting from direct and indirect force. Nontraumatic causes include (1) weakening of muscles due to (a) serious infections such as tetanus, typhoid, or influenza; (b) pregnancy, labor, and puerperium. (2) Severe arteriosclerosis, especially in elder persons. The case to be discussed falls into this latter category. Of interest are reports of this condition in young men during the influenza epidemic of 1917 and 1918.

Pathology consists of either a rupture of the muscle fibers with ensuing hemorrhage, or the rupture of one of the epigastric vessels or branches. This leads to infiltration of the muscle mass with blood, causing pain and spasticity that is hard to distinguish from the acute abdomen. There often is splinting of the muscles on the side of the hemorrhage that may be confused with the boardlike abdomen found in certain acute conditions. If hemorrhage increases, a mass appears which often is difficult to distinguish from an intraabdominal mass. In some cases, as in the one to be reported here, the mass may increase in size rapidly with a concurrent increase in area of pain, but there is no radiation of pain beyond the extent of the mass.

The authors feel that the syndrome of a rapidly enlarging painful mass in the abdominal wall

in the region of the rectus sheath is diagnostic of hemorrhage in the rectus sheath. A review of the literature concerning this entity reveals some illuminating points in prognosis and treatment. Reported cases have been treated both conservatively and by surgery with successes and failures reported in both instances. In a review of 107 cases, mortality of 7.5 per cent was reported[3]. This series included cases from the age of 3 months to 90 years, but other conditions may easily have led to this high mortality rate, since the average age of death was 60 years.

In the surgical treatment of this condition, preoperative diagnosis has ranged from acute empyema of the gall bladder (such as in our case) to any other intraabdominal emergency. At times the peritoneal cavity has been entered, complete examination performed, and then attention has been turned to the abdominal wall revealing the hematoma[4]. Therefore, incision over the mass at the time of surgery is strongly recommended.

The case presented in this report is that of a 73 year old white female, para 8, gravida 8, who is a hypertensive cardiovascular problem and also severely kyphotic due to ankylosing spondylitis. On the morning that she became acutely ill with vague abdominal distress that soon became severe enough to warrant notification of the attending physician, her chronic disease problem was static. She was on a management of hypotensive and cardiac drugs, Raudixin® and Diamox®, respectively, and was also on supplemental vitamins (Theragran-M®).

Just as this patient decided to get out of bed after awakening, pain started in the right upper quadrant over the gall bladder area. It was constant, severe, and aggravated by any attempted motion. Pain did not radiate but the painful area increased in size, extending from the right upper quadrant to the region of the umbilicus and inferiorly in a period of some two hours. Once it had reached two finger breadths below the umbilicus, pain became more severe and the patient resisted emphatically any attempt to change her position.

The original physical examination done approximately two hours after the onset of the acute abdominal episode revealed a blood pressure of 160/100, pulse of 90, and respirations of 16 per minute. Temperature was normal. She had compensated arteriosclerotic heart disease and kyphosis due to the long standing spondylitis. Generalized right sided muscle rigidity was noted with rebound to the right para-umbilical region. The examiner had the impression of an ill defined mass extending subcostally down to the umbilicus on the right. Rectal and vaginal examination were not helpful in deciding the nature of the acute episode.

In another hour, or by the time hospital admission had been accomplished, the mass had enlarged to two fingers breadths below the umbilicus and pain had increased in severity. The mass remained ill defined because of difficulty of accurate palpation caused by distress. The area of pain increased correspondingly with enlargement of the mass.

Laboratory work on admission to the hospital revealed a hemoglobin of 16 gm. or 100%, hematocrit 50, leucocyte count was 9,100 with 79% segs, 7% band, and 14% lymphocytes. Urinalysis, NPN, and blood sugar were within normal limits, as was the serum amylase. Prothrombin time and clotting time were normal. Platelet count was 379,000. Total protein was 5.9 gm. with an A/G of 1.95:1. Plain film of the abdomen revealed an ill defined soft tissue mass on the right side of the abdomen.

A tentative diagnosis was made of acute empyema or acute hydrops of the gall bladder. Since the patient's condition seemed to be deteriorating, it was decided to perform surgery with the plan of draining the gall bladder, using local anesthesia. Accordingly the patient was brought to the operating room and 1 per cent Xylocaine® was infiltrated throughout the line of incision. A transverse incision was made over the mass, extending from the midline laterally for four inches and midway between the xyphoid and the umbilicus. After the rectus sheath was opened, a considerable amount of clotted blood was noted and removed. This pocket of blood could be followed within the rectus sheath to below the umbilicus. In order to determine whether there was any intraabdominal pathology, a small incision was made in the peritoneum. Some serosanguineous fluid was noted in the abdomen, probably a transudate due to peritoneal irritation by the hematoma. Limited exploration of the abdomen by palpation through this incision revealed no pathology. The peritoneal incision was then closed.

A longitudinal extension was made inferior to

the original incision extending to the level of the umbilicus and bisecting the anterior rectus sheath. Exploration of this area revealed the clot to be infiltrating the muscle fibers, although the major portion of the clot extended inferiorly and posteriorly to the rectus muscle. As much clot as possible was evacuated and a small portion of the blood infiltrated muscle was removed for pathological study. No actual rupture of the muscle or bleeding point from the epigastric vessels could be demonstrated.

Penrose drains were placed into the depths of the wound and the incisions closed. The patient tolerated the procedure well and postoperatively she improved gradually. There was considerable drainage of dark brown material but no bright red blood. The drains were removed on the sixth postoperative day, but were replaced the next day because a pocket of fluid had formed. This drain was removed three days later and the wound healed well.

The pathological report was: "Sections reveal fragmentation of the (rectus) muscle fibers which show only beginning degenerative changes. However, between the fragments, masses of red blood cells are present and also polymorphonuclear leucocytes, the number of which is larger than could be expected if the presence of them would be due only to hemorrhage. Similiar hemorrhage also is present in the adjoining fat tissue. The blood corpuscles in general are quite well preserved and stained. In the sections examined, no source of the hemorrhage can be demonstrated, particularly no primary vascular lesion."

The microscopic diagnosis was: "Fairly recent hemorrhage, origin unknown."

SUMMARY

1. A case of spontaneous hemorrhage within the rectus abdominis sheath is presented.

2. This entity must be considered in the differential diagnosis of the acute abdomen, especially when

(a) pain is along the course of the recuts sheath,

(b) a mass is easily palpable, and rapidly increases in size, and

(c) the area of pain enlarges rapidly, with little or no radiation.

3. In case of doubt, incision should be made over the mass and the rectus sheath opened. This will avoid unnecessary opening of the peritoneal cavity.

4. Exact diagnosis often is established only by surgery.

REFERENCES

1. Hilgenreiner, H. as quoted in 3.
2. Beals, L.; Blanton, W.; and Eisendrath, D.: Abdominal Complications of Influenza Epidemic at Camp Custer, Michigan, J.A.M.A. 72:850 (Mar. 27) 1919.
3. Martin, D., Jr., and Thompson, E.B. III: Spontaneous Hemorrhage into the Rectus Muscle, Am. Surgeon 23:309 (Apr.) 1957.
4. Rogatz, T., and Rubin, I.L.: Hematoma of the Rectus Abdominal Muscle, New York J. Med. 54:675 (Mar. 1) 1954.

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Mental problems program

The first International Medical Conference on Mental Retardation will be held in Portland, Me., July 27-31. There will be speakers from Germany, England, Austria, and France as well as from many parts of the United States. Dr. Ella Langer, State House, Augusta, Me., is chairman of the Committee on Finance and Arrangements.

Pan-Pacific conference

The 8th Congress of the Pan-Pacific Surgical Association will be held in Honolulu, September 28-October 5. Nine surgical specialty sections will be held simultaneously. Information and brochures may be obtained from Dr. F. J. Pinkerton, director general of the Association, Alexander Young Building, Honolulu 13, Hawaii.

Treatment of Third Degree Burn with Tryptar Ointment

A. A. PALOW, M.D., KANKAKEE

Ordinary antibacterial agents, including antibiotics, frequently cannot penetrate the infected wound areas because they are blocked or inactivated by tissue debris. Reiser, et al.[1] have shown that topically applied trypsin and chymotrypsin rapidly digest tissue debris, but do not harm living tissue. Jenkins, et al.[2] reported on five cases of severe burns successfully treated with a trypsin-chymotrypsin debriding ointment. This same product, Tryptar® Antibiotic Ointment*, was used in this case. It contains crystalline trypsin and chymotrypsin to clear the burn of debris by proteolytic enzymatic action; and bacitracin and polymyxin to provide full antibiotic action to combat infection.

The patient, a 12 year old boy, sustained severe burns as a result of a gasoline fire. There were first and second degree burns over the entire anterior chest and abdominal wall, the medial and lateral aspects of the left upper arm, and of the medial aspect of the left forearm near the hand, and flash burns over the entire face.

TREATMENT

On being hospitalized, the patient was treated by debridement and pressure Vaseline® dressings to the burned areas. Skin grafts were begun two weeks later; all but one graft sloughed. Second grafts were attempted on the following lesions — left shoulder, right and left anterior chest walls, and medial and anterior aspects of the left upper arm. Ninety days later, the lesions on the left shoulder were approximately 5 centimeters in diameter; there were two lesions on the left chest 5 x 7 and 10 x 12 centimeters; one lesion on the right anterior chest wall 5 x 7

*Supplied by Armour Pharmaceutical Company, Kankakee, Illinois.

Before application of Tryptar Ointment.

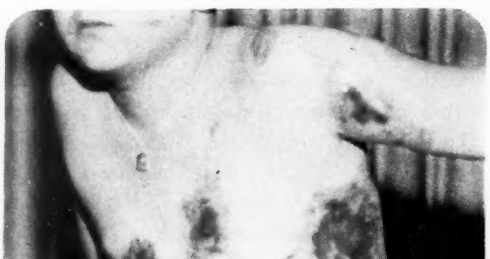
3 weeks after application.

5 weeks after application.

centimeters; the lesion on the left upper arm measured 7 x 9 centimeters. All these lesions were covered by thick granulation tissue with continual weeping of seropurulent fluids.

At this point, all lesions but one were treated with Tryptar Antibiotic Ointment. After one week, the dressings were removed. Changes in the treated lesions were so marked it was decided to treat all lesions with Tryptar Antibiotic Ointment. The dressings were changed at the end of seven days, when it was decided that daily applications of the ointment would be of greater benefit. The boy's mother was taught to apply the ointment and dress the wounds at home. The lesions had practically all epithelialized when the final inspection was made, approximately five weeks after treatment was started. As the dressings were done at home, only one visit a week to the physician was needed, resulting in a considerable saving of time.

Before application of Tryptar Ointment.



3 weeks after application.

5 weeks after application.

CONCLUSIONS

Although a considerable amount of ointment was used in this case, it should be remembered that this patient would have been subject to another skin graft, with doubtful results, since two previous attempts had failed. This also would have required another week to 10 days of hospitalization. Therefore, the use of this ointment has not only produced excellent therapeutic results but has greatly reduced the cost of the care of this patient.

REFERENCES

1. Reiser, H. C.; Patton, R., and Roettig, L. C.: Tryptic Debridement of Necrotic Tissue, Arch. Surg. **63**:568-575 (Oct.) 1951.
2. Jenkins, Ben H., and Powell, J. H., Jr.: New Concept in Management of Severe Burns. (Paper read before Coweta County, Georgia Medical Association.)

Clinical–Surgical Conferences

Diaphragmatic Hernia

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Discussants:
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PETER A. ROSI, M.D.
Associate Professor of Surgery,
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Dr. Robert Freeark: Few subjects in medicine remain as controversial and unsettled as the problem of diaphragmatic hernia. The internist and surgeon cannot often agree as to who needs surgery and the surgeons themselves frequently are at odds as to the best operative approach.

The vast majority of diaphragmatic hernias are through or adjacent to the esophageal hiatus. Their symptoms and complications involve structures on both the thoracic and abdominal side of the diaphragm and the multiplicity of problems produced makes the question of surgical interference a difficult one.

We are fortunate in having with us today two surgeons of outstanding ability and experience who are equally at home in the thoracic and abdominal cavities. I am sure they will have some strong convictions about the management of these cases. Dr. F. John Lewis is professor of surgery at Northwestern University Medical School with a background in gastronintestinal, thoracic, and cardiovascular surgery that is unsurpassed. Dr. Peter A. Rosi is associate professor of surgery at Northwestern University Medical School and for years has been one of the most esteemed members of the attending staff here at Cook County Hospital.

Case 1: Sliding hiatal hernia with acquired short esophagus. *Dr. Eugene Broccolo* (Surgical Resident): This 63 year old white male first entered Cook County Hospital in March 1956, complaining of pain in the epigastrium of two months' duration. It had been present actually for at least eight months but had been intermittent until two months prior to admission, when it became more frequent and began occurring at night. He obtained relief by sleeping in an upright position, by initiating vomiting, or by the ingestion of soda bicarbonate. He also had nausea after every meal, occasional melena, but no dysphagia or hematemesis. Appetite was good but he was afraid to eat because of discomfort. The patient's general health had been good. A right inguinal hernia had been repaired in 1954, and there was a history of excessive alcohol intake for 30 years but not for the immediate two years prior to this admission.

Case 1

At the time of his admission he weighed 164 pounds. Gastrointestinal study revealed the presence of a small hiatal hernia. A clinical diagnosis of duodenal ulcer was entertained and medical management instituted with some relief.

The patient was followed in the outpatient clinic and during the next two years was readmitted to the medical service on several occasions because of exacerbation of his complaints. In August 1956, he first became aware of dysphagia for solid foods and had lost 25 pounds. He was seen several times by the surgical consultant and surgery had been advised, but because of his fair response to medical management he was continued on therapy and watched carefully.

His present admission was for similar complaints with a recent episode of melena. Physical examination was not remarkable except for evidence of weight loss and mild anemia. His hemoglobin was 11.8 gm. and there were 85 clinical units of free acid in the fasting gastric aspirate. Barium studies demonstrated a short esophageal hiatus hernia with proximal esophageal dilatation and distal inflammatory changes. Esophagoscopy described hyperemic gastric mucosa 35 cm. from the upper teeth; the sphincter was patulous, the mucosa bled easily, and a gastric pouch extended up to a point where constriction was noted. The impression was of hiatus hernia with esophagitis, and the patient was transferred to surgery.

On June 4, 1958, through a transthoracic approach, relief of the condition was attempted. A tremendous reaction was seen around the esophagus so that the esophagogastric junction could not be determined accurately. Repair of the hernia was difficult and we were not satisfied with our efforts. We tacked the stomach up to the underside of the diaphragm and accomplished the best repair possible. The patient had an excellent immediate postoperative recovery with relief of dysphagia although he still has some abdominal pain.

Dr. Freeark: Dr. Meszaros will review the patient's X-ray films for us.

Dr. William Meszaros (Director of Diagnostic Radiology, Cook County Hospital): This first film, taken on November 29, 1957, shows considerable pouching of the stomach above the diaphragm. The esophagus opens into the stomach above the diaphragm, which makes it a sliding type of hernia. Narrowing and spasm of the distal esophagus suggest the presence of reflux esophagitis. Retrograde flow of barium from the stomach to the esophagus was easily demonstrated in the Trendelenberg position.

The next two films are dated June 24, 1958, which is a few days postoperative, and you see a small supradiaphragmatic pouch of barium which has the appearance of a small hiatus hernia. However, the amount of stomach above the diaphragm appears considerably less. The final film, taken on September 2, 1958, again shows the supradiaphragmatic pouch of barium. I believe the cardioesophageal junction also is above the diaphragm.

To summarize, this is a case of sliding hernia with esophagitis. On the final film the distal esophageal lumen looks wider and there is no radiologic evidence of esophagitis, but there still is gastric herniation through the diaphragmatic hiatus.

Dr. Freeark: We have a man with a two and half year history of symptoms compatible with a hiatus hernia. Throughout his two or three admissions to this hospital some question was raised as to whether this actually was the cause

of his symptoms. In time, he developed symptoms of complications of hiatus hernia, specifically esophagitis and possible stricture. Dr. Rosi, would you give us your opinion of the management of this case?

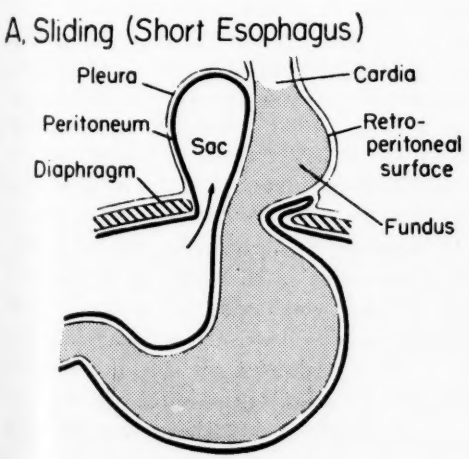
Dr. Peter A. Rosi: These symptoms result largely from interference with the function of the esophagogastric sphincter mechanism. This mechanism often is rendered incompetent in the presence of a hiatus hernia and thus permits the highly irritating gastric chyme to bathe the lower esophagus, producing regurgitant esophagitis.

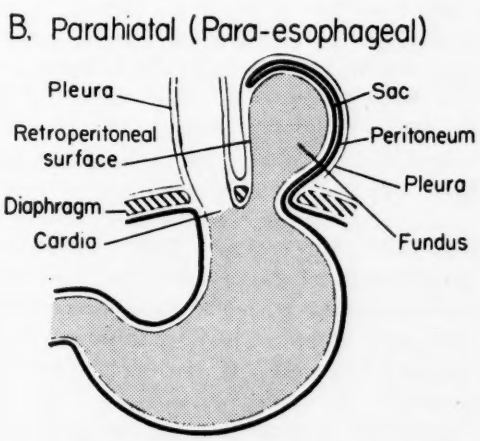
The nature of this sphincter mechanism is a matter of controversy. There is little doubt that in normal individuals such a mechanism exists to retain ingested food in the stomach despite the consumption of large volumes or the position of the patient. Anatomically, a collection of circular muscle fibers at the lower end of the esophagus is readily demonstrated. Yet this is hardly the muscle mass that characterizes other sphincter areas and it is felt that other mechanical factors—such as the oblique entry of the esophagus into the stomach or valvelike folds of gastric mucosa—act to prevent reflux. The slinglike arrangement of the diaphagmatic crura seems to play a part here also but regardless of the mechanism, competency is lost and herniation and regurgitant esophagitis occcur, as in the case presented.

A large series of these cases has now been reported and it seems that the aging process, with its associated lack of muscle tone, has something to do with it. As the diaphragmatic muscle sling around the esophagus becomes weaker the esophagus is permitted to come forward and the angulation between the esophagus and cardia becomes less, favoring reflux. Concurrently the muscle weakness permits the sliding hernia to become worse.

In doing a vagotomy, you are provided with an unusual opportunity to study the anatomy of this region. You can readily see the tremendous amount of protection at the esophagocardiac junction in the normal. The crural fibers come down around the stomach, protecting and holding it in the abdomen. With increased intra-abdominal pressure or with atrophy of the tissues, this protection weakens and hernia begins.

The big problem here is to decide which case is surgical and which is not. Most sliding hernias that do not produce reflux do not produce symptoms. Many times symptoms may be due to other causes as these hernias simulate upper abdominal or chest disease of various types. I don't know how to demonstrate whether a patient is suffering from sliding hernia or coexisting abdominal or thoracic disease. Some workers have injected air intraperitoneally and elevated the diaphragm and reduced the stomach into the abdomen, but I don't think the patient would like that. In the case presented an increasing esophagitis could be demonstrated due to reflux regurgitation of the acid gastric juices. There are two problems involved in treating this condition: Get the stomach back into the abdomen by repairing the hernia, and restore as well as possible the mechanism that prevents reflux. If there is hyperacidity, even mild in nature,





something may be done to correct it. Vagotomy and pyloroplasty are possibilities.

What about the surgical technique used in this condition? Many surgeons like to do them from above (transthoracic) while others prefer the abdominal approach. We prefer the latter for in most cases—and certainly with a very narrow constricted costal arch—you can get up close to the esophagus and work just as easily as by the other route. There are other advantages. If gallstones are present they can be taken care of, or you can reduce the total amount of acid production in a hyperacidity patient by some procedure on the stomach itself.

If bleeding or anemia has been a problem, a careful intra-abdominal exploration can eliminate other possible sources that might have been overlooked even with preoperative X-ray studies. In general the time required to open and close the abdomen is far less than a chest procedure and the recovery period, a lot more comfortable.

For these and other reasons we prefer the abdominal approach to almost all hiatal hernias. Repair of these hernias involves imbricating the crura behind the esophagus to hold the stomach in the abdomen. The esophageal hiatus is closed down enough so that you can just get the tip of a finger alongside the esophagus as it courses through the diaphragm. There is no agreement on how tight is should be. At first we made it quite loose but there were recurrences. Recently we have been making it much tighter and getting better results.

What about restoring the angle between esophagus and the stomach? Many times you can put a few sutures between the anterior wall of the stomach and the diaphragm to angulate the esophagus and stomach more acutely. Some have taken sutures between the esophagus and cardia but care is needed as they may tear out of the esophageal wall and cause perforation.

If the patient does not have hyperacidity, that is all you need do. If he has coexistent hyperacidity, you can offer him a great deal with vagotomy and pyloroplasty. Then, most of your patients will get good results. The group that has recurrences is an entirely different story, and they may have to have jejunal interposition operation for relief of the chronic type of esophagitis.

Dr. F. John Lewis: Do I understand correctly that there is still stomach above the diaphragm?

Dr. R. Freeark: We are very much afraid that that is true.

Dr. Lewis: Do you think this is a short esophagus?

Dr. Freeark: Yes. At surgery there was considerable doubt as to whether esophageal length would ever permit complete reduction.

Dr. Lewis: When you have a short esophagus almost always it is acquired. Sweet described four cases in a series of 111 which he thought were of congenital origin, and Allison thought there was one congenital short esophagus in a series of 250 cases of esophageal hiatal hernia. Congenital short esophagus, I think, can be thrown out of consideration. Most cases of acquired short esophagus seem to have esophagitis and ulceration, but there is no evidence of an active esophageal ulcer in this particular case.

When you are faced with the situation of an esophagus that will not allow you to pull the stomach down below the diaphragm into its normal position, then it is a perplexing problem. Some people feel you can always get it down through the hiatus, but there are cases—and this would appear to be one—where great difficulty is encountered. One solution is to transplant the esophagus to the dome of the diaphragm. This may shorten the required length considerably. If this is not successful in restoring the stomach exclusively to the abdomen, and it appears impossible, then you can stitch the stomach to the diaphragm and leave it above. The theory is that esophagitis is caused by obstruction, thereby allowing reflux; if you relieve this obstruction you can count on the sphincter action to get rid of the esophagitis problem.

It is my personal opinion that leaving the stomach above the diaphragm in some cases is a good solution. Another recommended procedure is to resect the entire area of distal esophagus and proximal stomach. This I do not think is a good solution; sometimes it is essential if there is real stricture at that point.

Dr. Freeark: This man tells us he is having a little abdominal pain. He is eating and has some difficulty swallowing. In general he is much improved since his operation. He says that he could neither eat nor sleep prior to surgery. Now I would like to ask Dr. Lewis for an expression of opinion about the surgical approach

to this type of hernia. Do you prefer thoracic or abdominal approach?

Dr. Lewis: My own preference is for transabdominal approach although some surgeons with more experience and of stronger character have shifted back and forth. In a few patients the transthoracic approach will be easier but the transabdominal approach gives you a better view of the crura, and a chance to deal with other pathology. It is quite evident that you can do the operation by either approach but there is some question whether you can do it consistently well by one as opposed to the other route.

Question: How long would you put up with this man's symptoms before you re-operated on him, or is this feasible? Obviously you have not completely solved the problem. Would you operate again or what would you advise?

Dr. Freeark: We like to think this particular patient is not greatly distressed by his postoperative symptoms. Certainly they are much milder than the complaints prior to surgery. This raises the question as to who must shoulder the responsibility for the failure of surgery to give complete relief. I am wondering if the persistence in medical management for over two years, with evidence of progressive herniation and esophagitis, does not present to the surgical team an almost insoluble problem. A scarred, shortened, and inflamed esophagus is certainly much harder to deal with than the relatively simple hiatus hernia with which his condition began.

Dr. Rosi: Preoperatively, if the patient is having symptoms of sliding type of hernia, he should have had surgery after a short course of medical management proved ineffective. He has never been overweight so the problem of weight reduction does not arise. If we assume that herniation remains or has recurred, keep in mind that operation next time would be of some magnitude. I would wait until the patient is very uncomfortable, so that his life is not quite bearable and he is willing to submit to another major procedure. The next surgical procedure I would entertain would be resection of the lower esophagus and the cardiac end of the stomach and interposition of a jejunal loop. To re-establish this continuity is a big order but it probably is the best approach.

Dr. Meszaros: If he has increasing symptoms would dilatation of the esophagus help?

Dr. Rosi: If he has increasing symptoms you have not overcome the etiologic factor. Dilatation of the esophagus would give some relief but he would still have regurgitation.

Dr. Paul Nora (Surgical Resident): If you were doing a cholecystectomy on a patient and you found a hiatus hernia would you fix it at that time, if the patient had had no previous symptoms that you could ascribe to the hernia?

Dr. Rosi: A great deal depends upon the patient's age. In a young individual you could repair it without trouble and probably forestall some symptoms. In an older individual without symptoms it is a questionable undertaking. I think you must consider why the patient is being operated; it may be that he is suffering partially from a sliding hernia as well as from gallstones. If he has had colic, he is suffering from gallstones and the hernia had nothing to do with it and should be ignored.

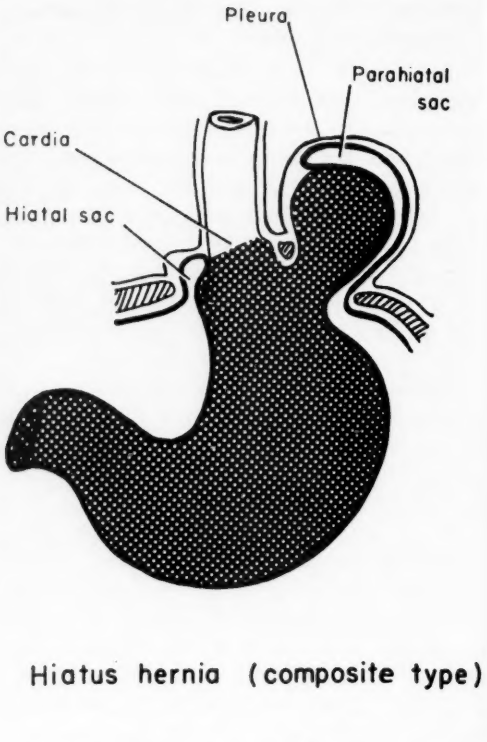
Case 2: Composite Type of Hiatal Hernia. *Dr. James Kane* (Surgical Resident): This 64 year old obese white female was referred to Cook County Hospital because of symptoms of anemia. She had been informed three months before admission that she was anemic but no diagnostic studies had been pursued. One week before admission she had a black stool on two occasions and noticed a moderate, dull, epigastric pain after eating. There also had been increased shortness of breath for the past few months. She had delivered five normal children. In 1948 she had a hysterectomy for carcinoma of the cervix. There is a strong family history of carcinoma.

On physical examination she was pale and obese. Temperature, respirations, and pulse were normal, and her blood pressure was 155/70. Her heart was enlarged, and there was a grade II apical systolic murmur. Abdominal and rectal examinations were negative, and the stool bensidine was negative. The patient denied nausea, vomiting, excesesive gas, indigestion, or prior episodes of abdominal pain.

Laboratory tests disclosed the following: Urinalysis negative. Hemogram: hypochromic anemia, hemoglobin 40 per cent, hematocrit 22, and RBC 3.5 million. Biochemical studies normal; total cholesterol 220. Electrocardiogram: left heart strain. X-ray films are on the view box.

Dr. Meszaros: These are all preoperative films.

Case 2



Hiatus hernia (composite type)

The first shows a faintly visualized gall bladder with many stones. She has a huge hiatus hernia superimposed on the heart shadow, and you have to rule out lung cysts and abscesses. Much of the stomach is above the diaphragm, and the esophagogastric junction must be above too. So we have a huge hiatus hernia with the esophagus coming into the stomach above the diaphragm. The esophagus does not look as short as the previous case. There is some tortuosity and the chances of pulling the stomach down beneath the diaphragm are much better than in the first case.

Dr. Kane: The patient was prepared for surgery; she was given 3,500 cc. of blood, and went to surgery on August 8, 1958. At surgery it was felt she had a composite type of esophageal hiatus hernia with some shortening of the esophagus and a paraesophageal hernia. Repair was from within the abdomen. Her recovery has been uneventful, and she was discharged to the clinic on the seventh postoperative day. (Patient shown here)

Dr. Meszaros: Would you define what you mean by a sliding, paraesophageal, and composite type of hernia?

Dr. Freeark: I understand that the composite or combined type has some features of both. What we are attempting to distinguish is the paraesophageal or parahiatal hernia in which the esophagogastric junction remains in its normal position at or below the esophageal hiatus and the bulk of the hernia occurs laterally or parahiatally to this location. In the sliding type, the esophageogastric junction slides above the hiatus and there is no interposed diaphragm between the herniated stomach and the esophagus. A combination hernia would be one in which both elements of herniation coexisted. The distinction often is difficult, and in this case there was some disagreement, even at surgery.

(On questioning the patient it was learned that she felt well and was having no difficulty at present; She had had some trouble swallowing solid foods during the second week after surgery but by the third week this had disappeared and she is eating well.)

Dr. Freeark: In contrast to the first case presented, this patient had a relatively short history. The main indication for repair here was the complication of bleeding. The transabdominal approach was used, and the gall bladder was

removed at surgery.

Dr. Lewis: Usually the paraesophageal type of hernia is not associated with peptic esophagitis and bleeding such as were found in this case. Many aspects of this woman's case fall into the classical picture. About two-thirds of these lesions occur in the female, most of them between the ages of 50 and 70 years. There is good evidence that the hernia gradually gets bigger. Blades defends the thesis that these hernias should be operated upon even when they are asymptomatic because many cause trouble as they grow. He had roentgenographic evidence in about 12 cases that the hernia grew in size over a period of years. I don't think most surgeons would agree that asymptomatic hernias should be operated upon. In many patients the condition is picked up incidentally; they remain symptom free for years.

Interest in these hernias has developed only in the last 10 to 15 years. Since this has occurred, better X-ray techniques have been used so more hernias are being diagnosed. The fate of a large number of asymptomatic hernias has not been worked out.

Except for the bleeding, this woman might have been considered neurotic because her symptoms were so vague. One difficulty in analyzing patients' problems is that neurotic tendencies may be present in addition to the hernia so their improvement or failure to improve with any management is dictated by psychologic reasons.

As for the success of operation, it troubles me to find that most surgeons who report their results have a recurrence rate of only 4 or 5 per cent, yet in my experience the rate is considerably higher. What is the recurrence rate? I don't know. If it is 10 per cent for inguinal hernia, it must be considerably higher for hiatal hernias.

It is my conviction that the technique of repair deserves further study and that the follow-ups should be as elaborate as the original studies. The situation in the first case, where the repair is really not successful, is quite common. If you have done any of this surgery you get the impression that it is not as nice and firm as repair of an inguinal hernia, for example. It is done with muscular tissue and little fascia or firm tissue is available to work with. You must remember that you are dealing with structures that move constantly and on the upper side of the diaphragm is a chamber with a constant negative pressure. This means that abdominal pressure exerts a greater relative force than in the inguinal region.

The immense sensitivity of the esophagus to acid has been mentioned. In experimental work, it has been shown that if you drip acid gastric juice on the cat's esophagus it will erode through within 6 to 8 minutes almost invariably. It is remarkable how sensitive it is. Many experiments have demonstrated that if you bathe the esophagus in acid-peptic juice you get esophagitis and perforation. A patch of gastric mucosa transplanted to the esophagus will cause ulceration of the esophagus. If the sphincter mechanism is lost it causes esophagitis but equally important is the erosive character of the gastric contents. It is a good thing the stomach empties through the duodenum rather than the esophagus. This consideration has led to development of the jejunal interposition operation mentioned earlier. I have had little experience with it and have no strong opinions of its use in this disease, but it deserves consideration where the risk becomes necessary.

The first patient presented here was approached by the transthoracic approach. That is the better approach in the short esophagus because you can free the esophagus up to the aortic arch and bring it down further and bring the cardia below the diaphragm. In spite of this approach, the desirable result was not obtained in that case.

Dr. Rosi: The second patient came in with melena and anemia. The question is, what caused this massive upper gastrointestinal bleeding. Is it due to diaphragmatic hernia and if so, when should it be repaired? Many times we operate upon these patients and bring the stomach down and the outer surface looks entirely normal without any evidence of the origin of eroded vessels. These bleeding points are undoubtedly from the mucosal side of the herniated pouch. In some patients if you look at the mucosa through an esophagoscope there occasionally is acute ulceration around the esophagogastric junction. At surgery the patient may have active bleeding from the cardiac end of the stomach. As you pull down the herniated end of the cardia into

the abdomen you may see engorgement of the veins in that particular portion of the stomach. In those patients you are sure you will get a good result. In others, it still remains a question whether you relieve symptoms or not.

Years ago some work was done on phrenicectomy in patients with bleeding and diaphragmatic hernias, and good results were reported. We did a few at that time but the patients continued to bleed a year or so later. They had excellent results for a year and then the condition recurred.

Dr. Lewis: I have not used phrenicectomy in these patients. There may be some indication for its use. Many cases are treated satisfactorily medically. Usually the ones I see demand more than phrenic crushing by the time surgery is acceptable.

Dr. Freeark: Recently a group has recommended the use of pneumoperitoneum as a nonoperative definitive measure. Have you any experience with that?

Dr. Rosi: No.

Dr. Lewis: No.

Dr. Freeark: We have postoperative X-ray films on the second patient.

Dr. Meszaros: Postoperative roentgenographic study showed that the hernia has been completely reduced. The stomach is completely below the diaphragm. There is some narrowing of the distal esophagus with some retention of food, indicating stasis.

Dr. Freeark: This stasis was at first symptomatic and has now disappeared without treatment. Dr. Rosi's conviction about a tight closure was adhered to in this case and we have no reason to regret it.

Dr. Freeark: How do you stand on concomitant vagotomy in this problem of sliding hernia? Do you practice routine vagotomy?

Dr. Lewis: If there is severe ulcer diathesis, vagotomy with pyloroplasty may be the best approach. Subtotal gastric resection has even been recommended. Some patients demand additional operation but what it should be I am not prepared to conclude. I have done gastric resection, but vagotomy may be better. Some of them will have duodenal ulcer with peptic esophagitis and perhaps an additional operative procedure to reduce acidity should be considered at the first operation. I really am not sure of the best course.

Question: How do you prefer to approximate the crura in the repair of a hiatus hernia, posteriorly or anteriorly?

Dr. Rosi: Posteriorly. This is important in reestablishing the esophagogastric angle.

Dr. Freeark: Have you ever transplanted the hiatus?

Dr. Lewis: Not recently, but I think I would still do it in case I could not get the stomach down below the diaphragm by other means. I am sure it is not as good a procedure as others but it may have a place in this condition.

EDITORIALS

Leukemia

Mortality statistics show a progressive rise in the incidence of leukemia since 1900. An estimated 11,000 new cases are observed in the United States each year. This increase cannot be accounted for adequately by the longer life expectancy nor by improved diagnostic methods.

The etiology of leukemia is unknown. An association between exposure to ionizing radiation and the subsequent development of the disease has been noted. Radiation appears to increase the occurrence of acute and of chronic granulocytic leukemia, but not of chronic lymphocytic leukemia.

Acute leukemia occurs almost exclusively in children and young adults. Chronic granulocytic leukemia is seen chiefly in the 20 to 45 year age group. Chronic lymphocytic leukemia occurs most frequently above age 55. The type of leukemia is diagnosed on the basis of cell morphology as seen in blood, bone marrow, and at times, tissue biopsies.

Since leukemia was first described in 1845, many agents have been found to alter the manifestations of this disease. Research on potentially effective substances continues at an intense level, and new experimental drugs are announced periodically. The drugs of choice today may well be discarded tomorrow. None of these agents has ever prevented or cured leukemia.

The selection of antileukemic therapy or the decision to defer specific therapy is determined by the type of leukemia, clinical manifestations, and hematologic changes. The agents used in the treatment of acute leukemia fall into two groups: the steroids and the antimetabolites. The steroid hormones (cortisone, hydrocortisone, corticotropin or ACTH, and prednisone,) in relatively small doses, modify the course of many cases of acute leukemia. In massive doses the steroids often induce complete clinical and hematologic remission. The antimetabolites include folic acid antagonists, purine antagonists, and glutamine antagonists. The most frequently used folic acid antagonists are aminopterin and amethopterin (Methotrexate®). Amethopterin is considered preferable.

6-Mercaptopurine (Purinethol®) is the drug of choice of the purine antagonists. 6-chloropurine and Thioquanine® do not appear to have any advantage over 6-mercaptopurine. The glutamine antagonists such as Azaserine and DON (6-diazo-5-oxo-L-norleucine) have no practical value when used alone in human leukemia. Whether they will be helpful in combination with 6-mercaptopurine is under study. Combinations of steroids and 6-mercaptopurine produce a 76 per cent complete remission rate in children while in adults the remission rate is 38 per cent. As a rule, more complete and prolonged remissions occur in children. The poorest results are obtained in patients over 50. Nonspecific measures of importance in therapy include transfusions for anemia, specific antibiotic therapy when in-

fection occurs, and platelet infusions for thrombocytopenia.

The agents that have been used in chronic granulocytic leukemia include X-radiation, radioactive phosphorus, urethan, Triethylene Melamine®, nitrogen mustard, demecolcine (Colmedie®), and busulfan (Myleran®).

In recent years Myleran has been used more extensively than any of the other agents in the therapy of chronic granulocytic leukemia. Results appear similar to those obtained from X-radiation. Bone marrow aplasia is the only serious toxic effect of this drug. Other side effects include amenorrhea, pigmentation of the skin, and gynecomastia.

Many patients with chronic granulocytic leukemia ultimately develop an acute exacerbation closely resembling acute leukemia. At this time 6-mercaptopurine and steroids may sometimes cause a remission.

Indications for therapy are more varied and the available agents are fewer in chronic lymphocytic leukemia than in chronic granulocytic leukemia. Chronic lymphocytic leukemia in the aged may run a benign course and have little effect on life expectancy. Generally, cases of chronic lymphocytic leukemia that do not appear to be progressive should not be treated.

Irradiation, nitrogen mustard, Triethylene Melamine, and chlorambucil (Leukeran®) are the principal forms of therapy for chronic lymphocytic leukemia. The steroids are mainly of value in controlling the secondary hemolysis and thrombocytopenia that occur in some patients.

Although there is still no cure for leukemia, in certain of these conditions, striking clinical and hematologic remissions can be produced. Many patients have been able to function for longer periods of time in their usual capacities, and life has been prolonged. The rapid rate at which new drugs are being discovered and tested should lead to the development of superior agents, if not cures, in the foreseeable future.

Louis R. Limarzi

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Polio has not been conquered

The campaign of immunization against paralytic poliomyelitis, conducted on a national scale over the past two years, is the most spectacular and successful battle ever waged against a disease. The incidence of polio dropped from a 5 year average of 35,321 cases from 1952 through 1956 to 5,894 cases in 1957, a decrease of 83 per cent. Only 5 per cent of the paralytic cases occurred in the most susceptible age group—children under 5 years of age—who had been protected by three doses of the vaccine. A 30 year battle against diphtheria, and a 100 year war against smallpox cannot claim results comparable to those obtained in a two year fight against polio.

Vaccination against polio has fallen short of its goals, however. Of the 110,000,000 susceptible people (under 40 years of age) in the United States more than 50 per cent are unvaccinated or incompletely vaccinated. The precipitous drop in the national incidence of polio in 1957 has been halted. In fact, the figures for 1958 show an ominous rise—a 30 per cent increase in paralytic cases over 1957.

There were serious epidemics in three areas in the United States last year. All occurred in low socioeconomic groups where the number of vaccinations was low. In all areas, the incidence of paralytic cases was highest in children under 5. Even in areas in which medical care is more adequate, the steep decline in polio was slowed or halted.

These disquieting facts are due mainly to two factors:

1. *Incomplete vaccination.* As the incidence of polio decreased, public apathy took over. Many families neglected to follow through on the vaccination series so zealously begun when the disease was rampant in their communities. As a result, in a discouragingly large number of instances three, or even two injections were not completed. The protection afforded by three properly spaced doses for all age groups is estimated at 87 per cent; for two inoculations, 74 per cent; for one, 35 per cent.

2. *Inadequate potency of vaccine.* The unfortunate experience in the early days of polio vaccination, when the safety of vaccine was inadvertently sacrificed for adequate potency, has been reversed too greatly. The present vaccine is undoubtedly free from active virus capable of producing the disease but increased safety is accompanied by decreased potency. This is being carefully and cautiously corrected. But our present low potency vaccine does not give permanent immunity. Protection begins to wane about a

year after the third injection.

It is squarely up to us physicians, and as important members of the team protecting the health of the people of Illinois, to collaborate in correcting these errors, and in realizing the goal of complete protection against polio. Here is what we must do:

1. We must see that *all* of our patients between the ages of 2 months and 40 years have two injections of polio vaccine one month apart, followed by a third injection five to seven months later. If possible, the third injection should be given before the polio season begins in July or August. However, there is no objection to giving any of the injections during the polio season. If an individual had one or two injections even a year ago, it is not necessary to start over, but only to continue with the second or third injection.

2. Because of the low potency of our present vaccine, a fourth injection is advised by all authorities. It should be given one year after the third. It restores active immunity within a few days, even if given several years after the initial series of injections. It is probable that this is a temporary expedient. With increasing potency of the vaccine, the duration of immunity from three injections will probably be prolonged greatly or indefinitely.

3. A review of the incidence of polio in the past year shows that the highest incidence of paralytic cases is occurring in areas of greatest congestion and of lowest income, and in children under 5. While urging vaccination for all under 40, we should concentrate on the areas and the groups in which polio flourishes, and from which it spreads. We must not limit our efforts to our own patients. We should work on a community level as vital members of a team made up of all available local resources—health departments, clubs, PTA, parent groups, schools, newspapers, and all other interested organizations in our area.

We must use whatever aids are available. Pharmaceutical houses are supplying placards for office display. The AMA has a series of mailing cards for notifying patients regarding their first three polio injections. They can be obtained through the Division of Communications, American Medical Association, 535 N. Dearborn St., Chicago 10.

Vaccine can be purchased through drug stores. A limited supply can be obtained free of charge through local health departments or through the Illinois Department of Public Health at Springfield. It is recommended that free vaccine be reserved for indigent patients. Through its local medical society, each community must work out its own method for reaching the indigent, the indolent, the complacent, and the uninformed.

In line with the unanimous recommendation of the House of Delegates of the AMA on Dec. 5, 1958, the Illinois State Medical Society and the Illinois Chapter of the American Academy of Pediatrics are sponsoring jointly a drive to conquer polio in Illinois. By action of the Council of the Illinois State Medical Society on Feb. 1, 1959, the secretary of each county medical society will be instructed to develop and implement a satisfactory program to meet the local situation.

Further information and advice may be obtained by writing to the Committee on Poliomyelitis of the Illinois State Medical Society, Monmouth, Ill., or to the committee of the same name of the Illinois Chapter of the American Academy of Pediatrics, P. O. Box 1116, Evanston, Ill.

John Lester Reichert

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Backward step

The American College of Physicians has joined Cutter Laboratories in appealing the decision of January 17, 1958 of the Superior Court in Alameda County, Cal., awarding two children, Anne Gottsdanker and James Randall Phipps, damages for polio infections allegedly resulting from the use of Cutter vaccine despite the jury's finding that Cutter was not negligent.

In its *amicus curiae* brief (friend of the court) it points out that "the creation of an absolute liability concept would greatly impair future progress. The introduction of new products and procedures would be stifled and mankind would be denied the continual advancement of medical science . . . . We believe that when, as in the cases before the court at this time, a biological is made according to strict government specifications and complies with the best scientific and productive knowledge available, and when the manufacturer is absolved of all possible negligence by the jury, as this defendent was, no liability should be incurred when an injury occurs because of the user's own peculiar suscep-

tibility or because of insufficient scientific knowledge at that time. To create such an absolute liability would be to saddle the world of medical science with an unfair burden . . . . . We in no way feel that we are overdramatizing these results for it is clear that researchers would be unwilling to try new drugs on patients, practicing physicans would be afraid to avail themselves and their patients of the new wonder drugs, and pharmaceutical houses would not be willing to manufacture new products should this concept be applied, for it holds the defendent liable without fault and liable for the unknown.

"Since the fact is self-evident that certain treatments will save lives or alleviate suffering, it is unrealistic and unreasonable to say that there must be no unknown untoward effects. If we take this position, then the conquering of disease in the future will be far slower, as neither manufacturers nor insurance companies can afford to insure against the unknown and the unpreventable. Thus, the lifesaving drug or biological that may save thousands of lives every year from cancer which might be available tomorrow would probably, under the absolute liability situation, be withheld for another ten years of testing and 'wait and see' and 'make sure' periods. To be sure, a statistically small number of hypersensitive or hypersusceptible individuals thus will be saved from harm, but in the meantime thousands who might otherwise live, or live without suffering, will necessarily be denied medical care.

"How can any scientist, physician, hospital, or pharmaceutical producer become involved in any forward steps in medicine, no matter how surrounded by standards, if he is to be held responsible for knowledge that does not, and cannot, exist until the future unfolds?"

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Dr. Ernest Edward Irons, AMA past president, dies

"The two unknown youths who committed this crime got only a handful of change—but they robbed Chicago of one of its great men."

This was the concluding paragraph of an editorial in the *Chicago Daily News* which paid tribute to Dr. Ernest Edward Irons, a past president of the AMA, whose death in the Presbyterian Hospital, Chicago, on January 18, at the age of 81 was a great loss to medicine and to the public which he had served so nobly in 55 years of practice.

Dr. Irons was knocked down by two thugs near his home on the night of November 19 and robbed of $1 in change. He never recovered from the injuries he received. Although a heart condition contributed to his death, a coroner's jury decided that the holdup injuries also were factors and returned a verdict of manslaughter. The assailants have not been captured.

Dr. Irons was born in Council Bluffs, Iowa, February 17, 1877. He obtained his bachelor of science degree from the University of Chicago in 1900 and his doctor of medicine from the Rush Medical College, Chicago, in 1903. He has practiced in Chicago since that year. The University of Chicago in 1912 awarded him the degree of doctor of philosophy for research.

From 1902 to 1904, he was assistant in pathology and bacteriology at the University of Chicago. Early in his career he became associated with the Presbyterian Hospital in Chicago. From 1923 to 1936 he was dean of students and dean of the faculty of his alma mater, where for many years he was clinical professor of medicine and chairman of the department. Since 1941, he held the title of (Rush) clinical professor emeritus at the University of Illinois College of Medicine.

In 1939, the Kappa Chapter of the Nu Sigma Nu Fraternity at Rush Medical College and at the University of Chicago established an annual lecture in his honor. In 1940, he gave the Charles R. Bardeen Memorial Lecture at the University of Wisconsin Medical School.

Dr. Irons was elected to membership on the Council on Pharmacy and Chemistry of the AMA in 1923, and served for 18 years. In 1929-1930, he was chairman of its Section on the Practice of Medicine and from 1940 to 1948 he was a Trustee. Following the death of Dr. Austin A. Hayden, Dr. Irons was elected to fill the unexpired term as secretary of the Board of Trustees. In 1941, he was elected secretary of the board, serving until 1948 when he was chosen president-elect. As president, and subsequently, he was a tireless worker in the campaign against socialized medicine.

He was a representative of the AMA in the World Medical Association. He was an ex-regent of the American College of Physicians, and in 1945, president; president of the American As-

sociation for the Study and Control of Rheumatic Diseases in 1934; of the Society of Medical History of Chicago in 1940; and of the Institute of Medicine of Chicago in 1946. He was a member of the Illinois State Medical Society, Chicago Medical Society, American Board of Internal Medicine, the American Rheumatism Association, Association of American Physicians, Central Society for Clinical Research, American Society for Clinical Investigation, and the American Association of Pathologists and Bacteriologists.

In World War I, Dr. Irons became colonel in command of the Base Hospital at Camp Custer near Battle Creek, Mich., and during World War II he represented medicine in many different capacities, including several important committees of the National Research Council.

In 1947-1948, he traveled abroad for the United States government to study health conditions. He went to Japan to recommend changes in that country's health programs for the post World War II occupation administration of Gen. Douglas MacArthur. He also make a study of medical practice in Puerto Rico.

Dr. Irons in 1947 was appointed by Mayor Martin Kennelly to the board of directors of the Chicago Municipal Tuberculosis Sanitarium his tenure there, Dr. Irons cut the institution's and he became president of the board in 1948. In death rate to one fifth of what it was in 1946. eliminated a long waiting list, and campaigned vigorously for more state aid. He was consulting physician at the Presbyterian Hospital. In 1946, he was awarded the "Goldheaded Cane" of the School of Medicine of the University of California. In 1958, he was presented the Chicago Medal of Merit for his contributions to the city, and the first annual Tuberculosis Institute Medal in 1958 for outstanding work in the field.

Distinguished for his contributions in medical research, especially in focal infection, iritis, and rheumatic diseases, Dr. Irons was accorded many honors by the medical profession. His recent writing on medical economics and social medicine have been extensive and authoritative.

Dr. Irons is survived by two sons, Dr. Edwin N., a staff physician at the Presbyterian Hospital, and Mr. Spencer E., an attorney, and six grandchildren. His wife, Gertrude, died in 1951.

Council meeting minutes

The regular February meeting of the Council was held at the Hotel Sherman, Chicago, Sunday, February 1, 1959, with the following present: Oldfield, O'Neill, Lorne Mason, Youngberg, Camp, Clark, Kirby, Hesseltine, Reichert, Portes, Piszczek, Dooley, Endres, Reisch, DuPuy, Goodyear, English, Montgomery, Fullerton, Hamilton and Reavley. Also present were: Percy E. Hopkins, Roland R. Cross, J. O. Firth, Walter C. Bornemeier, Wm. Schowengerdt, Everett Coleman, Paul A. Dailey, Louis R. Limarzi, James H. Hutton, Harry Mantz, Edwin Hirsch, F. M. Nicholson, George Turner, Richard J. Bennett; and Messrs. John W. Neal, Walter L. Oblinger, Albert Scott, and John A. Mirt.

Motion: (Fullerton-Piszczek) that the minutes of the December 14th Council meeting be approved as mailed to members. Motion carried.

Reports of officers

Dr. Oldfield reported as president, listing the meetings which he had attended since the last Council meeting. He reported that he had secured Dr. Frank L. Meleney from Miami University, Florida, to present the Address in Surgery; and Dr. Jerome W. Conn of the University of Michigan, Ann Arbor, for the Address in Medicine. He also has secured the speaker for the annual dinner, May 20, Miss Ann Landers, who will be the first woman to serve in this capacity and also the first woman to be seated at the speakers' table.

Dr. Camp distributed the financial statement and added that since the first of January, 1959, $169,046 in payment of dues had been received in his office. He stressed the importance of the counter secretary sending in all dues as they are received, and not holding them up until all physicians in the county have paid.

Dr. Montgomery reported as chairman of the Council. There was no official report from the Executive Committee, which had met on Saturday evening. The special committee has recommendations to make relative to a retirement plan for employees of the state society which will be presented at the March 8th meeting.

Advisory committee to I.P.A.C.

Dr. Montgomery reported as chairman of the

Advisory Committee to the Illinois Public Aid Commission. The Committee will appear before the commission in the near future and request an advance in fees for the physicians of this state.

Medical service and public relations

Dr. Hopkins, chairman, requested the floor for Mr. Oblinger, Mr. Neal and Mr. Scott.

Mr. Oblinger reported that the Legislative Manual was off the press and available for distribution throughout the state, and for the physicians attending the legislative meeting that same afternoon.

H. B. #6, developed by the ISMS, which provides for the testamentary disposition of the human body or parts thereof, has been introduced before the General Assembly. The bill is being sponsored by Mr. George Coutrakon and Dr. William A. Moore.

Two bills are being prepared to define the term psychiatrist as used in connection with the examination of persons charged with sexual crimes and to provide for the commitment and detention of those charged with being sexually dangerous.

A bill is being prepared to establish the definition of a privileged communication between a physician and his patient; and another bill to provide for the reasonable compensation of expert witnesses in court proceedings. Work on this latter bill is being deferred until the Committee on Industrial Health completes its suggestions along this line.

A bill has been prepared to define a professional person under the act requiring reporting of major visual limitations to the Department of Public Welfare.

Contact has been made with the Bar Associations to alert them relative to a joint program during the annual meeting, at the dinner to be held on Tuesday evening, May 19th.

The dinner for the members of the General Assembly will be held at the Leland Hotel, Springfield, on Tuesday evening, March 31st.

Mr. Oblinger introduced Mr. Albert Scott of Canton, associate counsel for the Illinois State Medical Society.

Committee on nutrition

Paul Dailey reported as chairman of the Committee on Nutrition. The joint conference on nutrition, sponsored by the committee of the ISMS and the Illinois Nutrition Committee, was held in Bevier Hall, at the University of Illinois, October 4, 1958. It was highly successful. There were 141 registrants (dietitians, nutritionists, physicians, home economics teachers, students, and faculty members of the University). The ISMS committee has been asked to sponsor another joint meeting in the fall of 1959 to be held at Western Illinois University in Macomb. It is the desire of the Committee on Nutrition of the ISMS that the Council grant the committee permission to carry on this worthwhile program. The committee at this time, desires permission to explore the possibility of participating in the proposed 1959 program.

MOTION: (DuPuy-Piszczek) that the Council grant the requested permission. Motion carried.

Journal committee and editorial board

Dr. Reisch reported that the joint meeting of the Journal Committee and the Editorial Board had been held on January 7. No special report will be made for the Council at this meeting. However, it is the suggestion that all members of the Council read over the recommendations of the Editorial Board (sent to all members by mail) and consider action at the next meeting of the Council. The duties of the Editorial Board should be more clearly defined, as well as how it is to function, and the rulings should come from the Council, since the publication of the Journal is one of its responsibilities.

Dr. Hirsch, as chairman of the Editorial Board, asked for Council guidance for future editorial board activity, and for an outline of responsibility and procedure. Dr. Montgomery stated that this question of responsibility for the editorial policy of the Journal and the duties and prerogatives of the Editorial Board had been a problem for a long time, and he would suggest that a committee of three be appointed from the Council to meet with the Editorial Board and formulate duties and bring recommendations back to the next meeting of the Council, to be made a matter of record. The committee appointed was Harlan English, Chairman, H. Close Hesseltine, and Edwin S. Hamilton.

Committee on narcotics

Dr. Reisch reported as chairman of the Com-

mittee on Narcotics. The committee met Sunday, January 25, in Springfield to consider possible changes, additions, and deletions to the Uniform Narcotic Drug Act. Present were Drs. Eli Borkon, G. S. Schwerin, J. E. Reisch as chairman, Mr. Walter Oblinger, and—by invitation—Mr. Malachi L. Harney and Mr. Paul J. Maton of the Division of Narcotic Control.

First consideration was given an amendment that would eliminate the necessity of pharmacists forwarding to the Division regular reports of Class B drugs sold on prescription. This amendment would bring the Illinois law into conformity with federal regulations.

Consideration also was given the treatment of the addict. The current law requires that the addict be treated in a county, state, or federal institution, and the proposed change would make it possible to treat the addict in certain private institutions (certain private sanatoria and psychiatric divisions of general hospitals). Mr. Maton suggested that the ISMS might be able to select the institutions or hospitals that would qualify, but the committee felt sure that the society would not choose to set itself up as an accrediting agency. It was suggested by the committee that the Department of Public Welfare already has standards by which psychiatric institutions and units of general hospitals are recognized and qualified, and the Commission might well work with this as a basis for certification.

It was the opinion of the committee that this change in the Narcotic Law is desirable, and the committee recommends that the ISMS work with the Commission to develop a plan whereby addicts may be treated in qualified private institutions.

The "search and seizure and penalty" (Section 23 of the Law) was discussed, especially the wording of this section and the penalty imposed. The Committee voiced objection to the phrase "without a warrant" but it was pointed out by the Commission that this same phraseology has existed in the Illinois law since 1937, and that so far as is known, it has not resulted in a single case concerning any physican; that this section of the law pertains primarily to the transportation of illegal narcotics; and that the section is in basic conformity with the federal law. The Commission further pointed out that this paragraph limits search and seizure activities to officers and employees of the Division, and does not give this authority to police officers or deputy sheriffs. They further stated that without the phrase "without a warrant" in the law, many of the Division's activities relative to shipment and distribution of illegal narcotics would be hampered seriously.

After prolonged discussion, the Committee felt that since this paragraph is not specifically applicable to a physician in the legal use of narcotics, and since it imposes no undue hardship or hazard on him, there is no basic reason for it to be changed. The Committee recommends, however, that the last portion of the paragraph, dealing with license revocation, be removed.

The Commission proposes to alter the current law by specifying that the prescribing of Class A narcotics in hospitals for hospitalized patients be accomplished by the writing of such orders on the hospital sheet. This procedure has been in use since the start of the present law, it being accomplished through the rules and regulations worked out by the Division and the Committee.

The Commission proposes to introduce legislation specifying a penalty of from one to three years' confinement for the unlawful taking, use, or possession of the official Class A prescription forms.

The committee suggested that the Commission might consider the personalizing of the official forms by imprinting thereon the name and address of the physician to whom they are issued.

The Committee considered and discussed the resolution submitted by the Sangamon County Medical Society, and also by other county societies. It was the opinion of the committee that the majority of the suggestions and changes called for by the resolution were already in the process of being accomplished, and that additional progress would be made in the treatment of the addict and instituted by both the Department of Public Welfare and private institutions qualified to treat the addict, as medical science advances and better treatment becomes available.

MOTION: (Fullerton-Piszczek) that the report be accepted. Motion carried.

Committee on nursing

Dr. Schowengerdt reported that the Committee on Nursing had met and considered the Belleville Junior College program. The commit-

tee feels that a meeting with the dean would be of assistance to learn exactly how the program is handled.

The committee recommends that the Society endorse the bill that would give the hospitals a subsidy of $400 per year per student nurse—a subsidy bill which we have not endorsed in the past. If state funds are not available, federal assistance may result.

Department of public health

Dr. Cross, Director of the Department of Public Health reported on several bills of considerable interest to the Department which will be introduced. (1) The Atomic Power Investigating Commission intends to introduce a bill to be known as the Radiation Protection Act. (2) On the recommendation of the State Advisory Board on Necropsy Service to Coroners (of which Dr. Hirsch is chairman) a bill will be introduced to establish a toxicological laboratory in the Department. It proposes an appropriation of $200,000. (3) An amendment has been proposed to the State Tuberculosis Sanitarium law which would authorize cancellation of charges against local governmental units that are entitled to subsidy payments and are indebted to the State for sanitarium service. (4) A bill to clarify access to official birth and death records may be introduced as an amendment to the present law.

Dr. DuPuy discussed the present birth certificates and the advisability of a society committee meeting with representatives of the division of vital statistics. By Council action the Chairman was authorized to appoint such a committee. Dr. Montgomery announced the personnel as follows: Newton DuPuy, Chairman, Jacob E. Reisch, and H. Close Hesseltine.

Committee on industrial health

Dr. Richard J. Bennett reported that his Committee had met January 20 with representatives of many interested groups. This group considered the question of workmen's compensation in Illinois. Research and meetings have brought out the fact that it would be impractical for the ISMS to introduce amendments to the Workmen's Compensation Act to obtain: (1) A physician as commissioner; (2) Two physicians on the State Council, and (3) Impartial Medical Testimony.

The one piece of legislation that has a good chance of being accepted would be to request a Medical Advisory Committee of five physicians who would meet once a month at no salary, and would make recommendations to the chairman of the Industrial Commission on matters brought to its attention with reference to medical problems only.

The members of the society committee, over a period of time, have talked to about 50 judges, 100 lawyers, and about 200 physicians about impartial medical testimony. Based upon the above information, the committee recommends that two recommendations be presented for approval:

(1) Pertaining to introducing legislation for an amendment to the Workmen's Compensation Act for a Medical Advisory Committee of five physicians.

(2) With reference to appointing a committee to select and set up Panels of Experts to be used in Federal and State Courts and for the Workmen's Compensation Commission.

These panels of experts will be available when they are requested to serve, and must be composed of men whose opinions are based upon extensive knowledge in the specialty involved. Their word must be unquestioned and they must function at each request. Dr. Hopkins suggested working with the deans of the medical schools, the ISMS, the CMS, and the Institute of Medicine to develop these panels.

MOTION: (DuPuy-Goodyear) that Dr. Bennett proceed with his work, and that the recommendations made by his committee receive the approval and endorsement of the Council. Motion carried.

Postgraduate medical education

Dr. Limarzi reported that the following postgraduate conferences are being set up: Olney on March 26; a panel on cancer at Mattoon on April 2; a panel on diabetes at Waukegan on April 8; and a miscellaneous program at Centralia on April 16. Lincoln has asked for a conference on March 19 but has not submitted its program requests.

MOTION: (Endres-Fullerton) that the suggested 15 minute health talks beginning in February over WJJD, Chicago, be approved, and that the Committee on Postgraduate Medical Education and Scientific Service assist in this work. Motion carried.

MOTION: (English-DuPuy) that a representative of the ISMS be sent to the AMA 14th Na-

tional Conference on Rural Health to be held at the Broadview Hotel, Wichita, Kansas on March 5-7. Motion carried.

MOTION: (Hamilton-Piszczek) that Mr. Oblinger represent the Society at the regional medicolegal conference planned by the AMA for Cleveland, Ohio, on April 3-4. Motion carried.

MOTION: (Hesseltine-Hamilton) that Dr. Richard J. Bennett represent the Society at the 1959 National Health Forum, Chicago, Palmer House, March 17-19. Motion carried.

MOTION: (Piszczek-Fullerton) that the following candidates for Emeritus and Retired Membership be approved. Motion carried.

EMERITUS: Apfelbach, George L., 6118 Sheridan Road, Chicago, CMS; Burket, Walter C., 1020 Lake Shore Dr., Chicago, CMS; Kaufmann, Gustav L., 2440 Lake View Ave., Chicago, CMS; Parker, William G., 1002 Main St., Mt. Vernon, Jefferson-Hamilton; Pearsall, Phebe P. Block, 1630 Fifth Ave., Moline, Rock Island; Petty, Ray H., 311 North Clay, Mt. Carroll. Carroll County.

RETIRED: Metzger, Hermann L., 6747 Bennett Ave., Chicago, CMS; Perlstein, Samuel, 1350 Lake Shore Dr., Chicago, CMS; Singer, Louis G., 520 South Burnside Ave.; Apt. 9J, Los Angeles, Calif., CMS.

Constitution and bylaws

Dr. Joseph O'Neill as chairman of the special Council committee, reported that the group was studying the proposed change in the constitution and bylaws presented by Winnebago County at the 1958 meetings of the House, as it pertained to the tenure of office of Councilors. The committee has written to all surrounding states, and also to several of the larger states to see how this matter is handled. The larger states contacted are California, New York, and Pennsylvania. On December 15 a letter was sent to all Councilors asking that each Councilor poll the delegates in his district for an expression of opinion relative to this question. We hope to have a detailed report for the March 8th meeting, and we hope all delegates to the meetings of the 1959 House of Delegates will be informed of the proposed change in the constitution and bylaws, and will have an opportunity to express the thinking of each county medical society.

The resolution pertaining to the reorganization of the Committee on Medical Service and Public Relations also is being studied and a special meeting will be held to go over the proposed changes carefully. We urge all Councilors to express the opinions of the county societies in their districts, and to keep the committee accurately informed.

Polio immunization for 1959

Dr. Reichert stated that the problem of polio immunization has not been solved; that much of the population is still without protection, and before the next polio season arrives, a campaign should be conducted to bring as large a segment of the population of Illinois within the safe limits provided by vaccination as soon as possible.

Dr. Reichert introduced Dr. Harry L. Faulkner, president of the Illinois Chapter, American Academy of Pediatrics, who spoke to the Council relative to the situation, and presented statistics available to the Academy.

NOTE: This material is not presented here as it appears elsewhere in the March issue of the Illinois Medical Journal in editorial form.

MOTION: (Reichert-O'Neill) that the Council sponsor a program for immediate vaccination against polio in accordance with the actions taken by the AMA House of Delegates as follows:

1. Each physician assumes the responsibility for making certain, whenever possible, that all members of families he serves receive protection against poliomyelitis by having the full three doses of polio vaccine.

2. State medical societies arrange with state health departments for a joint effort to bring together county medical society representatives and representatives of county and city health departments for the purpose of discussing the need for joint study committees at the local level to survey the problems which may exist and work jointly to solve them.

3. County medical societies meet with county and local health department representatives to create study committees to survey the problem of immunization as it may exist in the local area and develop and implement a satisfactory program to meet the local situation.

That this action consist of:

1. Publishing in the Illinois Medical Journal this motion, together with the facts on the present status of polio as given by Dr. Faulkner;

2. In notifying the secretary of each county medical society of the action of the Council, and instructing the secretary to work out the details of the local program on the basis of facilities available within his county.

Motion carried.

Discussion of the statewide alert followed with regard to informing the individual physician.

MOTION: (English-Reichert) that a letter be sent to every physician in Illinois giving him this information; and that another letter be sent to county medical society officers outlining local activity. Motion carried.

Dr. English stated that the University of Illinois has prepared a booklet on the management of emergencies that will be sent to every licensed hospital in the state if such a request is made to Dr. Warren Cole.

MOTION: (English-Goodyear) that such a request be sent to Dr. Cole. Motion carried.

Directory of licensed physicians

Mr. Neal called the attention of the Council to the fact that the new directory of registered physicians has been completed by the Department of Registration and Education and is now available. The department is mailing the directory to the state and county medical society officers and to all county clerks. If a physician's name does not appear, a letter to the Department will give current information. No additional supplements will be printed, but a new directory will be published each year.

The Council adjourned at 11:45 a.m.

Respectfully submitted,
HAROLD M. CAMP, M.D., Secretary

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Teamwork

Man, after something like 500,000 precarious years on this planet, has now succeeded in grasping firmly the reins of his own destiny. With this development come awesome responsibilities, in which no group shares to a greater extent than does the medical profession, unless it be the nuclear physicist, whose dawning appreciation of what he has done is so forcibly presented in Snow's novel, "The New Men." Who of us has not with pride identified himself with Sir Luke Filde's wonderful portrait of a physician keeping lonely vigil by the bedside of sick child? The physician's devotion to the individual patient is still unchallenged in a world of rapidly changing values. But, with signs that the human species as a whole may be confronted with medical problems no less serious than those of the child in Filde's portrait, who in this time of transition is keeping similar vigil over the species? We are all familiar with the complex apparatus of modern medicine, which briskly and efficiently swings into action when a patient with congenital heart disease enters the hospital for surgery, or a patient with an acute renal shutdown comes in for dialysis. But, in our concern for the individual, have we forgotten to set up the team that has as its concern the species as a whole? *James V. Neel, M.D. Medicine's Genetic Horizons. Ann. Int. Med, Aug. 1958.*

CORRESPONDENCE

Symposium on cancer in Chicago, April 16-18

The 18th Symposium on Cancer, sponsored by the American Cancer Society, Illinois Division, in conjunction with the Illinois State Medical Society, will be held in Chicago, April 16-18.

The symposium will place emphasis on early diagnosis and treatment and will consist of lectures, conferences and clinics.

The program follows:

Wednesday, April 16, morning at Northwestern University Medical School: "Tumors of the Lung and Mediastinum," Dr. Jerome R. Head; "Tumors of the Breast," Dr. T. Howard Clarke; "Tumors of the Kidney and Ureters," Dr. V. J. O'Conor; afternoon at University of Illinois: "Cancer of the Esophagus and Stomach," Dr. Gerald O. McDonald; "Tumors of the Larynx," Dr. Paul Holinger; "Carcinoma of the Lip and Jaw — Tumors of the Neck," Dr. Harry W. Southwick.

Thursday, April 17, morning at Mercy Hospital, Stritch School of Medicine of Loyola University: "Biopsy and Papanicolaou Smear," Dr. Charles J. Smith; "Carcinoma of the Female Pelvis," Dr. Herbert E. Schmitz; afternoon at Michael Reese Hospital: "Malignant Tumors of the Skin," Dr. A. M. Buchholz; "Carcinoma of the Colon and Rectum," Dr. Nathan Crohn; "Radiation Therapy of Malignant Tumors of the Breast and Cervix Uteri," Dr. Erich M. Uhlmann.

Friday, April 18, morning at University of Chicago Medical School: "Frontiers in the Use of Isotopes," Dr. Paul V. Harper; "Malignant Tumors of the Skeletal System," Dr. C. Howard Hatcher; "Cancer of the Bladder and Prostate," Dr. Cornelius W. Vermeulen; "Tumors of the Central Nervous System," Dr. John F. Mullen; demonstration of the Van de Graff generator, cobalt source, and linear accelerator, Dr. James W. J. Carpender.

Information may be had by writing to Dr. John A. Rogers, executive director, American Cancer Society, Illinois Division, 139 North Clark Street, Chicago 2.

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Clinics for crippled children listed for April

Twenty-four clinics for Illinois' physically handicapped children have been scheduled for April by the University of Illinois, Division of Services for Crippled Children. The Division will count eighteen general clinics providing diagnostic orthopedic, pediatric, speech, and hearing examination along with medical, social, and nursing service. There will be two special clinics for children with cardiac conditions, two for children with rheumatic fever, and two for cerebral palsied children.

Clinics are held by the Division in co-operation with local medical and health organizations, both public and private. Clinicians are selected among

private physicians who are certified Board members. Any private physician may refer to or bring to a convenient clinic any child or children for whom he may want examination or consultative services.

April 1 — Alton (Rheumatic Fever), Alton Memorial Hospital
April 1 — Hinsdale, Hinsdale Sanitarium
April 2 — Cairo, Public Health Building
April 2 — Flora, Clay County Hospital
April 3 — Chicago Heights (Cardiac), St. James Hospital
April 8 — Carrollton, First Baptist Church
April 9 — Springfield, St. John's Hospital
April 10 — Evanston, St. Francis Hospital
April 14 — East St. Louis, St. Mary's Hospital
April 14 — Peoria, Children's Hospital
April 14 — Quincy, Blessing Hospital
April 15 — Chicago Heights (General), St. James Hospital
April 16 — Elmhurst (Cardiac), Memorial Hospital of DuPage County
April 16 — Rockford, Rockford Memorial Hospital
April 16 — Watseka, American Legion Hall
April 21 — Belleville, St. Elizabeth's Hospital
April 21 — Danville, Lake View Hospital
April 22 — Elgin, Sherman Hospital
April 22 — Springfield (Cerebral Palsy), Memorial Hospital
April 23 — Bloomington a.m. (General), p.m. (Cerebral Palsy), St. Joseph's Hospital
April 23 — Mt. Vernon, Masonic Temple
April 28 — Effingham (Rheumatic Fever), St. Anthony Hospital
April 28 — Peoria, Children's Hospital

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Western cancer meeting

The annual Rocky Mountain Cancer Conference will be held in the Brown Palace Hotel, Denver, July 22-23. Guest speakers of national reputation will be on the program. A block of rooms at the hotel has been set aside on a first-come, first-served basis.

The conference will be sponsored by the Colorado State Medical Society and the Colorado Division of the American Cancer Society. Information may be had by writing to the Colorado State Medical Society, 835 Republic Building, Denver 2.

Proctology convention

The 11th annual convention of the International Academy of Proctology will be held in New York, April 5-9. The speakers will include specialists from the United States and foreign countries. Information may be had by writing to Dr. Alfred C. Cantor, executive secretary, 147-41 Sanford Avenue, Flushing 55, Long Island, N. Y.

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Nuclear medicine meeting

The 6th annual meeting of the Society of Nuclear Medicine will be held at the Palmer House, Chicago, June 18-20. For information, write to the Society, 750 North Michigan Avenue, Chicago 11.

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Symposium on VD

The 10th annual Symposium on Recent Advances in the Study of Venereal Diseases will be held at the Johns Hopkins Hospital, Baltimore, April 27-28. It is open to physicians and workers in allied fields interested in venereal diseases. The symposium is sponsored by the American Venereal Disease Association and the U. S. Public Health Service.

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AMA medicolegal meetings

The AMA will sponsor three regional medicolegal meetings this spring: Washington, March 20-21; Cleveland, April 3-4; Salt Lake City, April 17-18.

The program will include such subjects as medical and legal problems involved in narcotic addiction, traumatic neurosis, the approach of medicine and the law to contingent fees, *res ipsa loquitur* in professional liability cases, impartial medical testimony, and the classical method of examining an expert medical witness.

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Ophthalmology fellowships

Six additional fellowships for residents in ophthalmology will be awarded July 1 by the Guild of Prescription Opticians of America, Inc. Each fellowship is for $1,800, payable in monthly stipends over three years of residency. Information may be had by writing to the Guild, 110 East 23rd Street, New York 10.

Gastroenterology award

The American College of Gastroenterology has established an award for the best unpublished paper on research in gastroenterology or an allied field. The winning entry will receive $750 plus $250 for traveling expenses to present the paper at the annual meeting of the College in Los Angeles in September. Information may be had from the American College of Gastroenterology, 33 West 60th Street, New York 23.

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ICS meeting in Chicago

The 24th annual Congress of the North American Federation, International College of Surgeons, will be held in Chicago, September 13-17. The federation is composed of the United States, Canadian, Mexican, and Central American Sections. Write to the Secretariat, International College of Surgeons, 1516 Lake Shore Drive, Chicago 10, for information.

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Medical officers needed

Federal agencies have a wide variety of openings for medical officers, with salaries ranging from $7,510 to $12,770 a year. A brief description of the work and application forms may be obtained from the United States Civil Service Commission, Washington 25.

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Chest physicians to meet

The American College of Chest Physicians will hold its silver anniversary meeting at Atlantic City, June 3-7. The program will include papers, symposia, round table luncheon discussions, postgraduate seminars, and motion pictures.

Write to the American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, for further information.

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Oregon cancer conference

The Oregon State Medical Society, the Oregon Division of the American Cancer Society, the University of Oregon Medical School, and the Oregon Academy of General Practice will sponsor a cancer conference in Portland, July 16-17. There will be presentations by guest speakers from other parts of the country. A program may be had by writing to Mr. Roscoe K. Miller, executive secretary, Oregon State Medical Society, 1115 S. W. Taylor Street, Portland 5, Ore.

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Pediatric refresher courses

The Children's Hospital of Philadelphia and the Graduate School of Medicine, University of Pennsylvania, will offer refresher courses as follows: "Pediatric Advances," May 25-29; "Practical Pediatric Hematology," June 1-5. Inquiries should be addressed to Dr. Irving J. Wolman, Children's Hospital, 1740 Bainbridge Street, Philadelphia.

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O. & G. examinations

The American Board of Obstetrics and Gynecology will conduct oral and clinical examinations at the Edgewater Beach Hotel, Chicago, May 8-19. The deadline date for the receipt of new and reopened applications for 1960 examinations is August 1, 1959. Write to Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6, for information.

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Clinical pathology course

The Frank E. Bunts Education Institute, affiliated with the Cleveland Clinic Foundation, in conjunction with the Cleveland Society of Pathologists will offer a postgraduate course in clinical pathology in Cleveland, April 2-3. Information may be had from the Institute, 2020 East 93rd Street, Cleveland 6.

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Goiter meeting in Chicago

The American Goiter Association will hold its annual meeting at the Drake Hotel, Chicago, April 30-May 2. There will be papers and discussions dealing with the thyroid gland, its physiology, pharmacology, pathology, and therapy. Information may be had from the Society's secretary, Dr. John C. McClintock, 149½ Washington Avenue, Albany 10, N. Y.

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AAPS meeting in Texas

The Association of American Physicians and Surgeons will hold its annual meeting in Fort Worth, April 2-4.

Our civic duties

I think physicians should stop being afraid to speak out in public. Official government statistics tell us that there are some 5,000 chambers of commerce and some 10,000 luncheon and service clubs in America. There are thousands of voluntary and public groups of various types. One would assume that the average physician must belong to many of these different organizations. If so, it would seem that the physician would be highly informed on many facts of our political life and some of the special needs in the public health field. It would seem that by his very presence in these groups he should occasionally be so frightened or angry—or even so patriotic—to have long since roused himself in protection of his profession and his family and the public health from some of the threats that make their appearance there.

What's wrong? Why hasn't he? Perhaps the answer can be found in a survey that one state medical association conducted among its members as to their participation in public and civic affairs. Among other things the survey revealed that only 26 out of every 100 physicians belong to a chamber of commerce. Only 33 out of every 100 physicians were members of Kiwanis, Optimists, Elks, Rotary, or Lions. And only 15 out of every 100 physicians were serving on a board of health, board of education, as coroner, on the county board, county health committee, county public welfare committee, a city council or village board, or as mayor or alderman. Do these sound like the statistics of community leadership for which the medical profession has so great a reputation? *E. L. Bernhart, M.D. Drought in the Grass Roots. J.M.A. Alabama, Dec. 1958.*

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A cancer commission

A cancer commission could, at the state or local level, investigate new or notorious cancer remedies, publish their findings, and proceed according to the facts ascertained. If something apparently worthwhile materializes, the profession will be the first to study it—as it did in the case of insulin, the sulfa drugs, penicillin, and other now established agents. If, on the other hand, nothing worthwhile is disclosed, then the profession and the public should be informed. *L. H. Garland, M.B. The Pursuit of the Unorthodox. J. Michigan M. Soc. Apr. 1958.*

Postoperative diet

In the immediate postoperative period — for the first 24 to 48 hours after operation — there is inhibition of upper gastrointestinal secretions. After a brief period, these gastrointestinal secretions return to normal composition and normal volume. This is coincident with the return of gastrointestinal peristaltic activity as evidenced by the clinical examination. Since the secretions of the stomach, duodenum, pancreas, and biliary system total many liters a day, the internal secretion and reabsorption of these materials actually impose a greater physiologic burden on a gastrointestinal tract functionally than does any oral intake customarily given in the immediate postoperative period. It is well known also, that by the time ingested material is ready for evacuation from the stomach or the upper gastrointestinal tract, it is essentially a thick, smooth, homogenate of material with no coarse particulation. The adequate stimulus for at least part of the evacuating mechanism of the stomach is the provision of solid material, since it has been shown that a stomach may be distended even up to the point or rupture by liquid material without showing any change in intragastric pressure. It seems evident that the plan of providing patients with liquid material for the first two or three days of a postoperative feeding program is an erroneous conception of safety and a faulty interpretation of an adequate stimulus for gastric digestion and emptying. A complete reevaluation of the nutritional adequacy of feeding after surgery seems indicated. *Mark A. Hayes, M.D. Postoperative Diet Therapy. J. Am. Dietet. A. Jan.* 1959.

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Aspirin poisoning

An evaluation of the influence of candy medication on non-fatal aspirin poisoning is difficult. Data from the Chicago Poison Control Center indicates that, of the first 500 cases reported to the center, 84 were due to aspirin. Flavored "baby" aspirin was directly cited in 73 of the 84 cases — 14.5 per cent of the total cases received by the center and 87 per cent of the cases associated with aspirin ingestion. *R. A. Driver, M.D. Aspirin Poisoning in Children. J. Kansas M. Soc. June 1958.*

THE P. R. PAGE

John A. Mirt

Legislative battles on two fronts

Teamwork — which will require the united efforts of physicians, Woman's Auxiliary, medical personnel, and enlightened patients — will be necessary to protect the public against legislative moves that threaten the high quality of medical service being provided today.

This was the consensus at a legislative meeting, February 1, in the Hotel Sherman, Chicago, sponsored by the Committee on Medical Service and Public Relations of the Illinois State Medical Society. The meeting was attended by ISMS officers and councilors, county medical society officials and legislative chairmen from all parts of the state, Auxiliary representatives, and officers of medical assistants groups.

The medical profession this year is faced with threats on two fronts — Washington and Springfield. Legislators at the state and national levels in the first half of this year will consider numerous measures that will have a big bearing on the quality of medical care. Some bills will be good, others bad, from the public's standpoint.

It will be up to medicine to scan carefully all measures that concern health and to carry on an aggressive public education campaign for or against, as the case may require. Opinion must be mobilized, and legislators in Springfield and in Washington made aware of the thinking at the grass roots. There has been too much lethargy in the past. Continued complacency will mean more steps toward socialized medicine.

From representatives of the AMA, the legislative meeting learned that the principal threats in Washington are:

(1) A bill of the Forand type that will add social security beneficiaries to the already large group receiving federal medical care. This can be stopped only by offering a health insurance program covering the aged that will provide medical care at rates within the means of those over 65. The AMA and state societies all over the country already are working to develop something of that nature. The movement must be accelerated.

(2) Medicare will be the basis for an attempt in another direction to provide federal medical care. The appropriation in the current fiscal year for the treatment of dependents of service men through civilian sources was cut back to a level $21 million under expected requirements. This means more shifting of patients to military establishments, transfer of funds from other accounts, or deficiency appropriations.

(3) Service men's organizations are hard at work plugging for greater use of VA facilities for nonservice connected disabilities. The volume of this type of socialized medicine already is huge.

(4) It is quite possible that a federal employees health insurance program will be worked out, with the government carrying at least half the load. How this plan will be operated will have an important bearing on medicine.

(5) The so-called "doctor's draft" will expire on June 30. What the terms of an extension will

be is of utmost interest to the profession.

On the constructive side, the possible developments are:

(1) Legislation of the Jenkins-Keogh Bill type to encourage the establishments of pension plans by the self-employed. The measure passed the House last year, but became bogged down in the Senate. Prospects are more hopeful this year.

(2) Possible federal grants to medical schools. If this can be worked out through grants with no strings attached, medical education would be given a boost.

3) The extension of the Hill-Burton hospital construction program to nursing homes is being urged by the AMA. Support in Congress may be given.

At the state level, the most important pending program is a revision of the Medical Practice Act to provide better protection for the public. An interim commission has been hearing suggested amendments, and will submit recommendations to the Legislature. Illinois medicine has proposed a strengthening of the regulations against unlicensed medical practice by providing injunction powers to the courts and increased penalties. The foreign medical graduate problem also must be faced.

An example of constructive legislation proposed by the Illinois State Medical Society is House Bill No. 6. This would make anatomical material available through wills.

Among other constructive suggestions made by the state society are: (1) to amend the Workmen's Compensation Act so as to provide for the appointment of a medical advisory committee to the Industrial Commission; (2) to make patient-physician relationships privileged; (3) to eliminate the "bugs" which have developed in the Narcotics Control Act; (4) to correct the abuses in the subpoenaing of expert medical witnesses.

To obtain the enactment of helpful legislation and to combat effectively the measures which are a threat to the practice of medicine will require an energetic campaign from the grass roots up. This will mean that the individual physician must find time to devote to legislative matters; that the wives must carry the messages into every home in their community; that medical assistants must work hard to see that their friends are properly informed; and that patients must be made aware of how they will be affected by pending legislation.

Most important of all, the views of these people must be made known to legislators in Springfield and in Washington. This is no longer a "crash" program, but a long term project.

A blue print for this legislative action has been prepared for the Committee on Medical Service and Public Relations of the Illinois State Medical Society by Mr. Walter L. Oblinger, associate counsel. A copy of this "Legislative Manual for Committee Chairmen of County Medical Societies" may be had by writing to the Illinois State Medical Society, Monmouth, Illinois. Every person who has the health welfare of the public and the interests of the medical profession at heart should have a copy of this manual. It shows how to achieve results.

The AMA has reorganized its Washington bureau to carry on a long range program. This will reach all the way back to the county level. Field service will be provided.

In Illinois, forces are being mustered. The state society will carry on a broad educational program. Former State Senator Albert Scott of Canton, Illinois, has been employed as an associate counsel.

All these preparations will go for nought unless the individual physician, his family, his friends, and his patients are willing to give time and effort at the home level. In the final analysis, the future is up to the individual physician. He must be willing to be on the front line. It not only is a matter of better health care, but self survival as well.

Dr. Percy E. Hopkins, chairman of the Committee on Medical Service and Public Relations, was chairman of the legislative meeting. Other speakers were: Dr. Raleigh C. Oldfield, president of the ISMS; Dr. Walter C. Bornemeier, chairman of the Advisory Committee to the Woman's Auxiliary; Mrs. Charles W. Stigman, chairman of the Auxiliary Legislative Committee; Dr. Carl E. Clark, chairman of the Advisory Committee to the Illinois Medical Assistants Association; Mr. Warren Whyte of the AMA Law Division; Mr. John W. Neal, ISMS general counsel; Mr. Oblinger; Mr. Aubrey D. Gates, director of the AMA Division of Field Service, and Mr. Scott.

Leaflets on veterans' medical care

The AMA Council on Medical Service has

prepared three new leaflets explaining the medical profession's policy on veterans' medical care.

Each pamphlet covers a particular aspect of the problem. They show that the veteran loses by the continued expansion of this so-called free care and that there is an adverse effect on community medical care. The comparative cost to the taxpayer of local and VA medical services is brought out.

The council believes that these leaflets not only are helpful to physicians who plan to address lay organizations on this subject but of interest to physicians in general who may want clarification on this matter. Copies may be obtained from the AMA Council on Medical Service, 535 North Dearborn Street, Chicago 10.

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Commissural dermatitis

Commissural dermatitis, a dermatitic reaction at the corners of the mouth, is a common problem in elderly people. It has been thought that the erythema, scaling, and fissuring were manifestations of riboflavin deficiency but even with massive doses of riboflavin and other components of the B complex, the dermatitis persists. Although this type of dermatitis stems from a nutritional deficiency, the problem we are considering is not nutritional, but occurs as a result of anatomic changes in the aged. Inadequately compensated edentia, loss of elasticity, and loss of tone of buccal musculature are the anatomic changes that produce a fold at the corner of the mouth. In the presence of moisture, the two opposing skin surfaces become irritated, and an intertriginous dermatitis is produced. A seborrheic diathesis also may increase the propensity of certain old codgers to manifest this type of dermatitis. Once the inflammatory reaction is initiated, yeast and bacteria are superimposed. Treatment lies in the restoration of the anatomic defect by adequate dental prosthesis. In most instances, treatment designed to eradicate the specific infection (Mycostatin® ointment for yeast, tetracycline for bacteria) will improve the condition. In cases with a seborrheic element, a corticosteroid cream will result in dramatic improvement. *C. F. Burgoon, Jr., M.D. and J. S. Burgoon, M.D. Aging and the Cutaneous System. Geriatrics June 1958.*

The future of psychosurgery

In the past 20 years lobotomy and similar operations have been developed from crude beginnings through more and more extensive procedures — some so extensive it was found that if the patient survived he was ill-equipped to take his place in society again. While such procedures may produce occasional brilliant results in chronic schizophrenic patients, they are devastating to better preserved patients. Refinements in the matter of extent and in avoiding trauma to the cortex with its consequent danger of convulsive seizures, have made lobotomy a relatively safe operation. Observance of the proper indications and contraindications, and the proper timing of operations, has made it possible to return some 80 per cent of patients with a fixed state of tortured self-concern to a more cheerful and effective existence. When a patient is operated upon by one of the selective methods before deterioration has set in, the only personality changes that can be measured appear on the positive side of the ledger. This has been best shown in patients who were not in institutions, by Ayd, Gardner, Longo and his colleagues, and by me. With the present methods, such a rate of success cannot be attained in chronic patients in state hospitals. Nevertheless, psychosurgery may be on the threshold of another breakthrough that will bring another host of patients to better health. *W. Freeman, M.D. Psychosurgery. California Med. June 1958.*

AT THE EDITOR'S DESK

A PERENNIAL

Old stories don't die; they fade away temporarily but come alive periodically.

AP sent out a release from St. Paul, Minn., on Jan. 23, 1959 as follow:

[*This is a true story. Only the names have been omitted to protect the injured.*]

St. Paul, Minn., Jan. 23 (AP) — This is the story of a baby-sitter who really stuck to her job— and sat, and sat, and sat—far beyond the actual call of duty.

She was hired by a young couple in the Minneapolis suburb of Edina to watch their children while the couple attended a New Year's eve party.

In giving instructions to the 16 year old sitter, the lady of the house forgot one thing—that she had sprayed some of the bathroom equipment with a white plastic paint, the kind that "won't rub off, peel, chip, or blister. It stays on."

Early in the evening the baby-sitter visited the bathroom. When the young couple returned at 3 a. m., they found the perplexed, red eyed sitter still sitting there.

First the mother tried turpentine in an effort to unseat the unhappy girl, but she became convinced the paint was 100 per cent as advertised.

Then she had her husband call the family doctor, who came to the house at 4 a. m. He applied alcohol with no results.

"But," he assured the sitter cheerfully, "we'll find a way."

Despite the discomfiture of the seated sitter, the humor of the situation gripped the doctor. He burst into a spasm of laughter, tripped over the sitter's foot and hit the bathtub, knocking himself cold.

The young husband hastily called the emergency station at the hospital and within minutes an ambulance was on the way. The driver and attendant scooped up the doctor, who had suffured a concussion.

Then, with a hearty "allyoop" the ambulance crew tore the sitter from her sitting place and trundled her and the doctor to the hospital.

Both have recovered.

Attorneys later drew up two lawsuits against the young couple—one for the sitter and one for the doctor—but the suits will never be filed, an insurance company executive said Friday. They'll be settled out of court.

"We'd love the advertising we would get," the insurance man said. "But our concern for the parties involved is stronger."

"It's just too embarrassing all around."

The following story appeared in the Lancet more than six months ago.

Some twenty years ago, when seats were still substantially constructed in wood, a doctor and his wife went to a dance leaving their small child with a student as a sitter-in. On their return they could not find the young woman in

any of the ground floor rooms. Rushing upstairs they saw to their immense relief that the baby was sleeping peacefully; but strange noises were coming from the bathroom. The horrified doctor immediately remembered that he had varnished the seat that afternoon and had forgotten to mention it to their visitor.

When the door was burst open, it was at once clear that his fears had considerable substance. In his best professional manner, he reassured the sobbing and slightly hysterical girl and told her that her freedom was only a matter of moments. After two unsuccessful tugs he made a third very powerful heave, lost his footing, fell heavily to the floor, and broke his leg.

The situation was now serious; further professional aid was sought in the form of a plumber, who in his wisdom attempted nothing heroic but quietly unscrewed the seat. An ambulance was summoned and conveyed the doctor, the girl, and the seat to the local infirmary where they were admitted to the appropriate surgical wards.

According to an apocryphal postscript, the house surgeon felt he was too inexperienced to deal with such a complicated female case and left her for his chief's operating list in the morning. Eventually, the anaesthetized patient (seat in situ) was wheeled into the theatre, and the surgeon, turning to a postgraduate student notorious for his omniscience, said "Have you ever seen anything like this?" "Yes sir," was the reply, "Many times, but I have never seen it framed." *In England Now. Lancet Aug.* 16, *1958.*

PHARMACEUTICALS

Three hundred and seventy new pharmaceuticals were introduced in 1958 (400 in 1957). There were 44 new chemical entities in the group. Ayerst, Ciba, Lilly, and Merck-S&D, each brought three new chemicals; Abbott, Lederle, Pfizer, SK&F, Squibb, Upjohn, and Wyeth had two each. Kenacort (Squibb) and Aristocort (Lederle) appear to be headed for the million dollar class.

Drs. Jean Holowach and Don L. Thurston used Diamox to treat 56 epileptic children ranging in age from 3 months to 16 years. Of the 35 who experienced complete remissions, 19 had not had an attack for from 2 to 20 months at the time their cases were reported. Nine patients showed a 50 per cent improvement while 12 were not relieved. The rationale of the helpful effects has not been explained satisfactorily as yet.

Thioamide (alpha-ethyl thio-isonicotinamide) is a new drug with potentialities in tuberculosis treatment. It has been used on 78 patients by foreign physicians and was particularly effective in bringing about remissions. The product is believed to have its greatest value in treating cases of chronic pulmonary tuberculosis that have not responded to previous therapy when combined with isoniazid or isoniazid and streptomycin. However, the incidence of nausea and other gastrointestinal disturbances was so high, smaller doses had to be used.

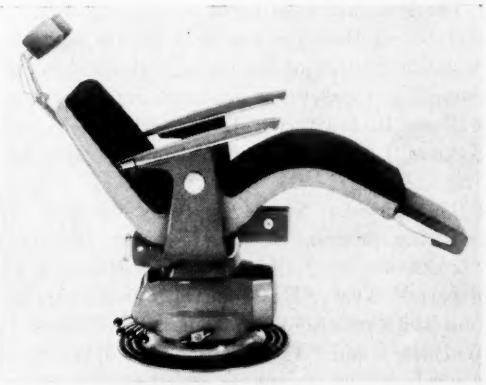
Singoserp is Ciba's new antihypertensive drug. It is a unique chemical development, as the reserpine molecule is split and then rebuilt.

B. M. Parker and associates reported some interesting observations on the use of the enzyme, Varidase (Lederle), in reducing the size of valvular vegetations in experimentally produced endocarditis in dogs.

Sebulex, a new therapeutic shampoo, is marketed by Westwood Pharmaceuticals. The product is aimed specifically at itchy, scaly scalp with a tendency toward dryness. It contains a mixture of anionic surface-active cleansers and wetting agents.

DENTAL COMFORT

The Ritter Company has designed a comfort-



able new dental chair that has been engineered to "cradle the patient in a semi-reclined position during dental procedures."

NEWS of the STATE

ADAMS

SOCIAL. The February meeting of the Adams County Medical Society was their annual social event with music by Junior Musolino and his "Cardiac Six."

COOK

MEETINGS. The February meeting of the Society of Medical History of Chicago had the following program: "Hermann Boerhaave and the Rise of Clinical Teaching", by Steven Armentrout; and "Antyllus, Surgical Genius of Antiquity," by Richard L. Grant. Both speakers are on the faculty of the School of Medicine, University of Chicago.

The February meeting of the Chicago Society of Internal Medicine was held jointly with the Scientific Section of the Chicago Heart Association. The speakers at the meeting were Drs. William B. Wartman; Emmanuel Marcus and William P. McKeever; and Peter V. Moulder, Jr.

The Chicago Neurological Society had the following program at their February meeting: "Treatment of Narcolepsy with Ritalin," by Robert E. Yoss; "Experimental Cerebral Infarction and Anticoagulant Therapy," by Jack P. Whisnant; and "A Review of Glossopharyngeal Neuralgia," by Joseph G. Rushton.

Dr. Robert E. Cooke, professor of pediatrics, Johns Hopkins Hospital, spoke on "The Effects of Sodium on the Central Nervous System," at the February meeting of the Chicago Pediatric Society.

ANNUAL LECTURE. The annual D. J. Davis Memorial Lecture will be given at 1:00 p.m., Apr. 22, Room 221 of the Dental-Medical-Pharmacy Building of the University of Illinois, 1853 W. Polk St., Chicago. The lecture will be "The Early Development of Internal Medicine in the United States," by Dr. Lloyd G. Stevenson, professor of the history of medicine and dean of the faculty of medicine, McGill University, Montreal.

NEW POSTS. Dr. J. W. Henry has been elected medical staff president of St. Francis Hospital, Evanston, where he served as chief pathologist since 1948.

Dr. Herbert Bessinger, University of Illinois College of Medicine faculty member, was appointed director of medical education for Louis A. Weiss Memorial Hospital.

Dr. J. L. Keely has been appointed professor and chairman of the department of surgery at the Stritch School of Medicine of Loyola University. He was appointed also head of surgery at Mercy Hospital.

Dr. Jordan A. Hunt was elected medical staff president of MacNeal Memorial Hospital, Berwyn.

Dr. Herbert C. Pollack, practicing clinical roentgenologist, Chicago, has been named chairman of the art committee of the International College of Surgeons' Hall of Fame.

Dr. Edward R. Pinckney was appointed direc-

tor of the comprehensive medicine clinic at Northwestern University Medical School. He is also in charge of preventive medicine and public health teaching, and will serve as executive secretary of the committee for an integrated program for education in medicine at Northwestern.

Dr. Edmund F. Foley, professor of medicine, University of Illinois School of Medicine, was appointed president of the staff at Cook County Hospital.

SERIES OF LECTURES. Michael Reese Hospital Medical Center lists the following lectures which will conclude their present series on "The Management of Medical and Surgical Emergencies," given by their hospital staff members: Apr. 1, "Common Poisonings in Children and Adults," Arthur H. Rosenblum and Edward A. Newman; Apr. 8, "Status Asthmaticus," Milton Mosko and "Hemoptysis" and "Respiratory Failure: Mechanical Respirators," Gordon L. Snider; Apr. 15, "Massive Gastrointestinal Hemorrhage," Charles H. Lawrence and "Hepatic Coma," Mitchell A. Spellberg; Apr. 22, "Tetany, Thyroid Storm, and Addisonian Crisis," Rachmiel Levine and Morton Smith; and Apr. 29, "Acute Abdominal Emergencies," Samuel L. Goldlberg and "Acute Urologic Emergencies," Irving J. Shapiro.

NEW LABORATORY. The Comdr. Eugene F. McDonald, Jr., Memorial Laboratory for Exfoliative Cytology has been established at the University of Chicago Medical Center. It was financed by leaders in the radio and television industry as a memorial to the late founder-president of Zenith Radio Corporation of Chicago. Dean Lowell D. Coggeshall said Comdr. McDonald and his friends had been interested in research seeking to expand the use of exfoliative cytology. The new laboratory includes a suite of five rooms located on the second floor of the Mothers' Aid Research Pavilion at the Chicago Lying-In Hospial on the University of Chicago campus. The laboratory is designed to make examinations for gynecological, lung, and other forms of cancer routine. The staff of cytologists works under the leadership of Dr. George L. Wied, assistant professor in the department of obstetrics and gynecology.

HONORED. Dr. John A. D. Cooper, professor of biochemistry and assistant dean of Northwestern University Medical School, had the degree of Doutor Honoris Causa conferred upon him by the University of Brazil in recognition of the work he has done in helping to establish and equip a radioisotope laboratory in the Institute of Biophysics at the Brazilian medical school.

Awards for 30 years of service to Michael Reese Hospital's Medical Center were presented to Dr. Otto Saphir, director of the department of pathology, and Dr. Joseph Greengard, attending physician in pediatrics.

RETIREMENT. Dr. Eleanor M. Humphreys, associated with the department of pathology of the University fo Chicago since 1926, has been made professor emeritus and consultant. For 11 years prior to retirement Dr. Humphreys was in charge of surgical pathology.

POSTHUMOUS AWARD. By unanimous action this month, the Board of Chancellors voted to bestow posthumously the Gold Medal of the American College of Radiology upon Dr. Warren W. Furey, who died suddenly in Chicago last November, while attending the 44th annual meeting of the Radiological Society of North America.

MEETING. Dr. William J. Grove, associate professor of surgery, University of Illinois College of Medicine, spoke on "Indications for Cardiac Surgery" at the Jan. 27 meeting of the DeKalb County Medical Society.

KNOX

MEETING. Dr. Charles B. Puestow, clinical professor of surgery, University of Illinois College of Medicine spoke on "Biliary Diseases" at the February meeting of the Knox County Medical Society.

LAKE

MEETING. Members of the Lake County Medical Society held a discussion on the free choice of physician, the closed panel systems, and social security for physicians at their February meeting.

LASALLE

MEETING. Dr. Stephen E. Reid, assistant professor of surgery, Northwestern University Medical School, spoke on "Unusual Lesions of the Right Colon" at the February meeting of the LaSalle County Society.

OGLE

NEW OFFICERS. The 1959 officers for Ogle County Medical Society are: Drs. Clyde L. Dren-

nen, president; Thomas E. Cunningham, secretary-treasurer; and L. Thomas Koritz, publicity chairman.

PEORIA

MEETING. Dr. T. R. Van Dellen, Chicago spoke on "Trials and Tribulations of a Medical Columnist, " at the February meeting of the Peoria Medical Society.

RICHLAND

POSTGRADUATE MEETING. A postgraduate conference sponsored by the ISMS Postgraduate Medical Education and Scientific Service Committee will be presented at the Richland Memorial Hospital, Olney, March 26. Four members of the faculty of the University of Illinois College of Medicine will present the program.

A luncheon at the Richland County Country Club will be followed by talks at the Hospital on "Pediatric Emergencies," by Dr. Heyworth N. Sanford; "Refractory Heart Disease," by Dr. Jacob W. Fischer; "Treatment of Acute Hand Injuries," by Dr. John H. Schneewind; and "Gynecologic Sexual Deviations," by Dr. James H. McClure. There will be question and answer periods.

A fellowship hour will precede the dinner, at which the speaker will be Dr. Harlan English of Danville, councilor for the eighth district. Physicians from all surrounding counties are invited to attend.

ST. CLAIR

MEETING. Dr. Oglesby Paul, clinical associate professor of medicine at the University of Illinois College of Medicine was the speaker at the February meeting of the St. Clair County Medical Society.

SANGAMON

MEETING. Dr. William Requarth, Decatur spoke on "The Treatment of Traumatic Wounds," at the February meeting of the Sangamon County Medical Society.

VERMILION

MEETING. Mr. George C. Mahle from the Danville School Board talked briefly on the coming school bond issue at the February meeting of Vermilion County Medical Society. The speaker of the evening was Walter L. Oblinger, ISMS associate counsel whose subject was "Operation Legislation."

WASHINGTON

NEW OFFICERS. The Washington County Medical Society elected the following 1959 officers: Drs. C. W. Longwell, president; K. W. Eirich, vice president; and W. P. Plassman, secretary-treasurer.

WHITESIDE

NEW OFFICERS. Whiteside County Medical Society elected the following officers for 1959: Drs. L. J. Milcarek, president; Jack Alter, vice president; and T. E. Flynn, secretary-treasurer.

GENERAL

ACCREDITATION. The office of Governor William G. Stratton announced that Peoria State Hospital has received full accreditation by the Joint Commission on Accreditation of Hospitals. This is for one year, or until re-inspection.

Information has been received from Miss Greta Jones, researchist for Reader's Digest, that enhances Peoria State Hospital on a national scale. In preparing an article, not yet published, on the "open" hospital movement, research revealed that this was the first completely "open" hospital in the United States. It was designated an "open" hospital in 1908 under the superintendency of the late Dr. George A. Zeller. Miss Jones defined a completely "open" hospital as one having all of the wards and residential units unlocked for a period of a minimum of eight hours a day.

PUBLIC HEALTH NURSING. The School of Nursing at the Chicago Professional Colleges of the University of Illinois has been awarded full accreditation for its public health nursing program by the National League for Nursing, Department of Baccalaureate and Higher Degree Programs. Miss Emily C. Cardew, dean of the School of Nursing, said the school is one of two in the state to receive accreditation in public health nursing. The public health program consists of 12 weeks of study for senior students. They receive their training in Peoria from the city and state public health departments and the Peoria Visiting Nurses Association.

TRAUMA SECTION. The United States Section, International College of Surgeons, has formed the Section on Surgery of Trauma as a successor to the Section on Occupational Surgery. Dr. Chester C. Guy, clinical associate professor of surgery at the University of Illinois College of Medicine is chairman of the section and Dr.

N. Gillmor Long, Evanston and Chicago, is co-chairman and secretary.

A New Radio Series Over Radio Station WJJD

The Illinois State Medical Society has been invited by Radio Station WJJD to give a series of recorded radio programs as a public service presentation on the last Wednesday of each month from 6:40 p. m. to 6:55 p. m.

John L. Reichert, assistant professor of pediatrics at Northwestern University Medical School, and a member of the Chicago Board of Education, presented the first program, FEBRUARY 25, on "The Conquest of the Inexcusable Diseases."

LECTURES ARRANGED BY THE ILLINOIS STATE MEDICAL SOCIETY:

GEORGE M. CUMMINS, associate in medicine, Northwestern Unversity Medical School, addressed the Loyola Golden Age Club of the Jewish Community Centers, March 18, on "Factors in Keeping Healthy."

LECTURES ARRANGED FOR THE ENGLEWOOD BRANCH OF THE CHICAGO MEDICAL SOCIETY:

February 3, GEORGE F. LUTTERBECK, professor of radiology, Cook County Graduate School of Medicine, "Practical Use of Radioisotopes."

March 3, ARTHUR W. FLEMING, associate professor of pediatrics, Stritch School of Medicine of Loyola University, "The Premature Infant."

April 7, HARRY A. OBERHELMAN, professor and chairman, Deparment of Surgery, Stritch School of Medicine of Loyola University, "Management of Carcinoma of the Colon."

PAUL R. ROSENBLUTH, clinical instructor in neurological surgery, University of Illinois College of Medicine, Stock Yards Branch of the Chicago Medical Society, April 17, on "Recent Management of Parkinson's Disease."

GEORGE E. KIRBY, Councilor of the Second District of the Illinois State Medical Society, Sheffield Woman's Club in Sheffield, April 1, on "Mental Health."

DEATHS

CORRECTION: The January issue of the Illinois Medical Journal carried an incorrect report of the death of Dr. William G. Sachse of Morris as a result of information from his county medical society. We are happy to announce that Dr. Sachse is very much alive and is still interested in a Fifty Year Club membership. We regret the error.

HANS C. ARON*, Chicago, who graduated at Friedrich—Wilhelms—Universitat Medizinische Fakultat, Berlin, Prussia, in 1908, died December 11, aged 78. He was certified by the American Board of Pediatrics.

RICHARD C. BENKENDORF*, Bushnell, who graduated at the Chicago Medical School in 1936, died January 8, aged 50. He was a member of the American College of Chest Physicians, and the American Trudeau Society.

GEORGE F. BLOUGH*, Odell, who graduated at the University of Illinois College of Medicine in 1903, died January 22, aged 87.

WILLIAM H. CUNNINGHAM*, retired, formerly of Rockford, who graduated at Chicago College of Medicine and Surgery in 1909, died recently, aged 77, at the home of his daughter in Pittsburgh.

JOSEPH NORMAN ELLIOTT*, Bloomington, who graduated at Northwestern University Medical School in 1921, died January 21, aged 64. He coached freshman football at Northwestern University during World War I, and later at Illinois Wesleyan. He was certified by the American Board of Otolaryngology.

ALLAN H. FERGUSON*, who graduated at the Hahnemann Medical College and Hospital, Chicago, in 1913, died January 19, aged 74. He was past president of the medical staff at Illinois Masonic Hospital, and assistant professor of gynecology and obstetrics at the Chicago Medical School.

WILLIAM B. FONVIELLE*, Rockford, who graduated at Howard University College of Medicine, Washington, D. C., in 1928, died December 31, aged 60. He had practiced medicine in Rockford since 1934 except for two years, from 1943 through 1945, when he served as medical officer in the United States army.

WATSON GAILEY*, Bloomington, who graduated at the University of Illinois College of Medicine in 1904, died January 19, aged 76. In 1931, at the invitation of the British army, he spent two months in northern India removing cataracts from the eyes of the natives. In 1941, he built the Gailey Eye Clinic in Bloomington. He attended the First International Congress on Corneal Transplantation of the eye in New York City in 1949, and in 1951, the Watson Gailey Eye Bank, the sixth of its kind in the United

*Indicates member of the Illinois State Medical Society.

States, was set up at Mennonite Hospital. He was a member of the American Academy of Ophthalmology, and the Chicago Ophthalmological Society.

CHARLES H. HAMILTON*, Vermont, who graduated at Eclectic Medical College, Cincinnati, in 1894, died recently, aged 91.

FRANK W. GRIFFITH*, Elgin, who graduated at the Chicago College of Medicine and Surgery in 1907, died January 19, aged 78. He was a member of the staffs at St. Joseph and Sherman hospitals in Elgin.

MILTON C. HANDELMAN*, Oak Park, who graduated at the University of Illinois College of Medicine in 1933, died February 5, in Hollywood, Fla., while on a vacation. He was 51. A member of the staff of Mount Sinai Hospital since 1937, he was also associated with the Weiss Memorial Hospital.

HENRY B. KNOWLES*, Anna, who graduated at St. Louis University School of Medicine in 1911, died January 24, aged 74. An employee of the Illinois Department of Public Welfare for 38 years, he served in the Peoria State Hospital before coming to Anna. He was superintendent of the Anna State Hospital from 1939 to 1941.

HERBERT R. KOBES*, Springfield, who graduated at Harvard Medical School, Boston, in 1930, died January 10, aged 54. He was associate professor of pediatrics at the University of Illinois College of Medicine, and Director of the Division of Services for Crippled Children at the University of Illinois.

EDMUND G. LAWLER*, Chicago, who graduated at Loyola University School of Medicine in 1925, died January 23, aged 58. He was assistant clinical professor of pediatrics at Stritch School of Medicine of Loyola University, head of the pediatric department of Little Company of Mary Hospital, a member of the American Academy of Pediatrics, and the American College of Physicians.

JACOB MYERS, retired, Sherman Oaks, Cal., formerly of Chicago, who graduated at Rush Medical College in 1905, died January 19, aged 75. Until his retirement in 1953, he was an attending physician at Woodlawn Hospital, and attending surgeon at the Spalding School for Crippled Children.

ROBERT D. PERKINS*, Moline, who graduated at Rush Medical College in 1935, died December 31, aged 50. He was a member of the American Urological Association, and the American College of Surgeons.

DUANE W. PROPST*, Oak Park, who graduated at the University of Illinois College of Medicine in 1919, died February 3, aged 67.

JOHN H. REEVES*, Chicago, who graduated at Bennett Medical College in 1896, died January 14, aged 84. He had practiced medicine in Chicago over 60 years.

OTTO F. SEIDELMANN*, Flossmoor, who graduated at the Chicago Medical School in 1941, died January 29, aged 44. He was a member of the staff of St. James Hospital in Chicago Heights, and formerly was assistant attending ophthalmologist at Children's Memorial Hospital.

IRWIN D. SIMINSON*, retired, formerly of Chicago, who graduated at Rush Medical College in 1924, died in his home in Mineral Point, Wis., January 26, aged 64. He was former chief surgeon for the New York Central Railroad in Chicago.

JOSEPH B. SONNENSCHEIN*, retired, Chicago, who graduated at Rush Medical College in 1902, died January 30, aged 82. In 1906, he joined the staff of the Chicago Board of Health where he served as supervisor and clinician of the department's venereal disease control division. In 1957, the University of Chicago Medical Alumni Association honored him with a testimonial dinner for his 55 years of work in medicine.

HENNING M. SWENSON*, Chicago, who graduated at the University of Illinois College of Medicine in 1929, died January 17, aged 58. He was a member of the surgical staff of Augustana Hospital.

MATTHEW TAUBENHAUS*, Chicago, who graduated at Medizinische Fakultat der Universitat, Wien, Austria, in 1928, died January 16, aged 55. He was vice chairman of the Department of Medicine and research associate at Michael Reese Hospital.

LOUIS J. TINT*, Chicago, who graduated at Jefferson Medical College of Philadelphia in 1908, died February 4, aged 80. His avocation was color photography, and he was noted for his landscape photographs of places around the world.

*Indicates member of the Illinois State Medical Society.

Angina plus

In a series of 238 patients with angina pectoris, other significant associated pathology was present in 113 (48.3%). Sixty-nine (29%) patients found at least a 50 per cent diminution of the frequency of their pain following successful treatment of the associated pathology. Nineteen patients (8%) were not improved. Twenty-five (11%) were lost to follow-up. The results of this study indicate that all patients having angina pectoris deserve thorough investigation of extracardiac factors that might be contributing to the frequency and severity of their attacks. Frequently one or more such factors can be found and corrected, and the patient's clinical condition improved even though the fundamental abnormalities in the coronary arteries are unaffected. Congestive heart failure, obesity, hypertension, and gallstones have been the most frequent of such contributing factors. *A. L. Cornish, M.D. Contributing Causes of Angina. J. Kentucky M. A. May 1958.*

Coronary sclerosis and goiter

Postmortem findings in 250 persons who died of coronary sclerosis are compared with the findings in 250 persons who died from other causes.

Goiter was commoner, and the average weight of the thyroid was higher, in those with coronary sclerosis. Among the coronary sclerosis cases, those with goiter had a tendency to lower average age at death and a higher average heart weight. Comparison of body build showed that the 250 persons with coronary sclerosis were slightly shorter and heavier than the controls. Among those with coronary sclerosis, those with goiter were heavier than those without goiter.

These findings are correlated with observations made in Finland on the nature and distribution of endemic goiter, the iodine content of the food, and the distribution of coronary sclerosis.

It is concluded that goiter and arteriosclerosis may have a pathogenetic relationship, possibly through hypothyroidism and the overproduction of thyroid-stimulating hormone by the pituitary gland. *Unto Uotila, M.D. et al. Goiter and Arteriosclerosis. Lancet July 26, 1958.*

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Hibernation

According to the theory of hibernation the life of a deeply shocked and normally homeostatic animal can be saved only by blocking the homeostatic reactions, thereby preventing the onset of extreme exhaustion. Hibernation achieves this by a multiple neural block, a controlled inhibition of the neurovegetative system. By suspending homeostasis, hibernation reverts the organism to a poikilothermic pattern. Life processes slow down. Twilight condition and hypometabolism set in. Hypothermia is an associated feature; even without cooling it will occur as a result of hypometabolism.

Hibernation corrects vascular collapse by closing the sphincters and by eliminating the engorgement of the capillaries, of the arteriovenous anastomoses, and of the metarterioles. This mechanism is essential in treating shock by hibernation. In prevention of shock an additional advantageous feature is the reduction of the basal metabolic rate. Hypothermia by cooling also leads to a reduction of the basal metabolic rate, but it does not offer the stabilizing mechanism of the peripheral vascular circulation. *A. Strelinger, M.D. Clinical Experience with Hibernation. J. M. Soc. New Jersey June* 1958.

Congenital defects

A description of events during the first 12 weeks of pregnancy and of other maternal characteristics was recorded for 3,295 women at their first prenatal examination. Seventeen were found not to be pregnant, and 62 could not be traced. The remaining 3,216 pregnancies resulted in 68 spontaneous abortions, 74 stillbirths and neonatal deaths (excluding those with major congenital defects), 50 congenital defects classed as major and 72 as minor, and 2,987 apparently normal infants. There were 119 premature births (excluding those associated with major defects, multiple births, and induced labor).

Mothers of the 72 infants with minor defect and of the 119 premature infants did not differ in any respect from the mothers of normal infants and so were included with these in subsequent comparisons.

As compared with mothers of normal infants, a higher proportion of those of infants with major defects had an acute febrile illness or pulmonary tuberculosis or had been engaged in certain types of heavy work in early pregnancy. Women who aborted also had an excess of acute febrile illnesses but not of the other factors. Stillbirths and neonatal deaths were associated with a raised incidence of acute illness, anemia, and vaginal blood loss. Mothers in all three abnormal groups had had an increased rate of fetal loss in earlier pregnancies. *A. D. McDonald, M.D. Maternal Health and Congenital Defect. New England J. Med. Apr.* 17, 1958.

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Steroids in tuberculosis

The greatest difference of opinion regarding the place of steroids exists in the field of tuberculosis. Some have felt that this form of therapy is always contraindicated while others have recommended its use under certain specific circumstances. Some of the tissue damage and clinical manifestations in tuberculosis are due to an exaggerated interaction between sensitized tissue and tuberculoprotein. Corticosteroids may suppress this overactive defense mechanism with a resulting decrease in the manifestations of illness. In patients seriously ill with tuberculosis of long duration there is evidence of adrenocortical hypofunction. Steroid therapy used with concomitant antituberculosis chemotherapy often effects striking symptomatic improvement. Thus, without anticipating any change in the ultimate outcome, the use of steroids would appear to be justified, if only for its symptomatic effect, in patients hopelessly ill with advanced tuberculosis. In acute forms of tuberculosis associated with severe clinical illness, steroids may be helpful. This is especially true of miliary and meningeal tuberculosis. In the latter condition, prevention and relief of cerebrospinal fluid block has been attributed to steroids. *Present Status of Chemotherapy in Tuberculosis. J. Indiana M. A. June* 1958.